Vol. 5 No. 1. September, 1945

# ANNALS OF THE RHEUMATIC DISEASES





CONTENTS



LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

YEARLY SUBSCRIPTION (4 NUMBERS) 25/- U.S.A. $6.00 SINGLE NUMBER 7/6

## NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1.

## NOTICE TO CONTRIBUTORS

Papers submitted to this Journal are accepted on the understanding that they have not been and will not be published in any other journal, and are subject to editorial revision. All papers and other editorial communications should be addressed to Dr. C. W. Buckley, 5, The Square, Buxton, Derbyshire.

The author of an original article should make adequate references to previous work on his chosen subject.

A full summary of his observations and conclusions must be given.

A paper describing a single case will not be accepted unless the case is sufficiently rare, or shows important features not previously described, or has been made a subject of special observation or experiment.

Articles must be as concise as possible and be typewritten on one side of the paper only, with double spacing and a margin of not less than 1¼ inches. Only recognized abbreviations should be used. Graphs, charts, tables, and legends for them should be presented on separate sheets and not included in the text. When half-tone reproduction of x-ray illustrations is required, the author is advised to send the original film unless he wishes to bring out special points in a print of his own choice. Photographs and photomicrographs should be printed on glossy paper, should be larger than the size desired for reproduction, and, if transmitted through the post in a tube, should be rolled with the picture outside. With the exception of letters and numbers, which should be lightly written in pencil, everything that is to appear in the reproduction of a graph or chart should be carefully drawn in black ink on tracing linen, or Bristol board, or stout, smooth, white paper.

References should be arranged according to the Harvard system. In the text, the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (*a*, *b*, *c*) after the date. No numbering of references is necessary. At the end of the contribution references are arranged in the alphabetical order of the author's names. The reference details are given as follows: Author's name and initials, year of publication (in parentheses), title of periodical (in italics, abbreviated according to the *World List of Scientific Periodicals*), volume number (bold type, Arabic numerals), and first page number (ordinary type, Arabic numerals), thus:

Cowan, J. (1929). *Quart. J. Med.*, **22**, 237.

When a book is referred to, the full title, publisher, the place and year of publication, edition and page number should be given.

Contributors will receive one proof in page, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of ten shillings per sheet of sixteen pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

Fifty free reprints of articles will, if desired, be given to contributors. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of costs will be given on application to the Publishing Manager, British Medical Association.

Papers which have been published become the property of this Journal, and permission to republish must be obtained from the Editors.

Application for Advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

c
w
w
o
h
st
sk

o
st
al
st
si
b
m
d
a
c
It
th
si
d
o
th
th
id
q
sy
tr
al

fr
ir
m
st
re
a
th
sp
tr
th

# THE NATURE OF FIBROSITIS

## I. THE MYALGIC LESION AND ITS SECONDARY EFFECTS: A REFLEX THEORY

BY

MICHAEL KELLY

*Perth, Western Australia*

Hench and his collaborators (1941a, 1941b) have complained more than once of the dearth of original work upon fibrositis. Nearly all of those who write upon the subject, they say, merely copy the observations of Stockman (1920); and, of those who have attempted to make original pathological studies, the work has been too inadequate and sketchy to be of much value.

### Should Demonstrable Pathological Changes be Expected?

To many writers, the term fibrositis bears a degree of reality corresponding to the extent of the demonstrable pathological change in the tissues. If abnormal thickenings and nodules can be demonstrated in the painful areas, the diagnosis of fibrositis is accepted; but the case is suspect if nothing but tender spots can be found. There has been much debate on the subject, the doubters often demanding organic proof before they will believe, and the believers mostly asserting that such proof could be provided were a biopsy but practicable. It has seemed to the present writer, however, that the case has been argued incorrectly from both sides. The doubters are not entitled to demand demonstrable changes in the tissues as a condition of acceptance; and it must follow therefore that the believers should not find it necessary to assert their faith in the reality of such changes. For the idea of pain unaccompanied by organic change is quite familiar to all, and in many recognized painful syndromes, usually called neuralgias, the idea of trying to find structural alterations has long been abandoned.

#### THE PAINS OF FIBROSITIS

The pains of fibrositis, however, frequently differ from the more discrete painful states in their irregular distribution and in the generally vague manner in which the patient describes them. Instead of being localized to a well-defined spot or region, they are often spread irregularly over a large area; they may not be made worse by movement; there may be no objective signs or only a few tender spots in the deep tissues. The pains may be intractable, and wearing both to the patient and to the physician; it is small wonder that they so frequently acquire the label "psychogenic." But those who believe in the existence of fibrositis, as a somatic rather than as a psychic disturbance, would do well to remember that there is no evidence to support their opponents in their demand for objective changes. The "myalgic spots," which have been demonstrated in the majority of cases by a large number of observers, are in themselves enough to explain all the symptoms.

#### ASCERTAINED PATHOLOGICAL CHANGES

Stockman quotes Balfour, in 1816, as the first to describe definite thickenings in chronic rheumatism; since then their existence has been confirmed many times. Stockman described swelling or necrosis of collagen fibrils, associated with collections of round cells and surrounded by an oedematous or fibrinous reaction. In the more chronic cases fibrosis of the tissues supervened. Collins (1940) removed some subcutaneous thickenings and found only normal fatty tissue with some fibrous encapsulation; he examined Stockman's material and was convinced that many of the sections showed little departure from the normal. Abel, Siebert, and Earp (1939) reported low-grade inflammatory changes with early degeneration of muscle-fibres; in their chronic cases fibrosis was observed, with degenerative changes more advanced. In the cases reported by Jordan (1941) the painful and indurated tissues did not appear abnormal upon microscopy. More recently Copeman and Ackerman (1944) explored the backs of a number of sufferers from lumbago, and found the main lesions to consist of fatty tissue with proliferation of fibrous tissue and some vascular congestion. Travell, Rinzler, and Herman (1942) appeared to strike a true note when they summed up as follows: "It is possible that the fibroblastic proliferation which has been observed is secondary to a functional disturbance and occurs only if the latter persists for a period of time."

Hench and his collaborators have suggested that a useful beginning could be made by an intensive study of the fibrous and muscular tissues at all ages; this would indicate with certainty what are the normal results of ageing and of prolonged use. Until this is done, they say, we have no data upon which to base our ideas of the "abnormalities"

found in fibrositis. In the opinion of the writer even the most comprehensive study would prove inconclusive, and in a proportion of undoubtedly painful areas no histological change would be observed. The changes associated with rheumatic disease are closely related to the changes inherent in normal degeneration, and there always will be some cases in which the observable changes are so early or so mild that they are not easily differentiated from the normal.

## The Myalgic Spot and its Effects

The myalgic spot is a small area of tenderness; but it sometimes give rise to a larger area of secondary tenderness of a less acute nature. Emphasis was laid upon this distinction by Kellgren; and in the experience of the writer it is an important distinction. The myalgic lesion is recognized by its more acute tenderness, and by its affinity for certain well-defined "sites of election." The smaller lesion is the cause of the diffuse radiating pain, sometimes accompanied by patches of secondary tenderness. Upon the injection of procaine into the lesion, both the radiating pain and the secondary tenderness will be observed to disappear forthwith. This result is diagnostic; if it is not obtained, the injection has not been correctly placed. The phenomenon suggests a few questions relating to the nature of the secondary tenderness.

### ARTIFICIAL MYALGIC LESIONS AND SECONDARY DEEP TENDERNESS

Experimentally produced deep somatic lesions cause not only radiating pain, but "referred" tenderness of the deeper structures in the painful area (Lewis, 1938; Inman and Saunders, 1944; Granado, 1945). All are agreed that the tenderness appears at the same time as the pain, and abates when the pain abates. It must follow that the tenderness is a reflex phenomenon; the time-relations can be explained upon no other basis. When the primary lesion ceases to give off painful impulses, the surrounding deep tissues cease to be tender to pressure. This deep hyperalgesia, therefore, must be the result of reflex nervous impulses, acting in a fashion analogous to the reflex production of cutaneous hyperalgesia (Lewis, 1937, 1942). Lewis demonstrated that cutaneous hyperalgesia could be produced by the anti-dromic stimulation of sensory nerves; its spread in the skin from a point of injury he showed to be brought about by the medium of axon-reflexes. In the efferent component of an axon-reflex, the impulses run antidromically in the nerve-fibre and release a "pain factor" at the nerve-endings.

Arguing from analogy, it seems reasonable to regard the appearance of deep hyperalgesia as the result of a similar mechanism in the deep tissues. The myalgic spot behaves like the artificial lesion, sending off a succession of abnormal impulses which cause reflex effects in the deep tissues. A small amount of procaine solution, correctly placed, cuts off the stream of impulses and causes the immediate abatement of the secondary tenderness. Deep hyperalgesia, which is the result of a neuro-chemical change, should not of necessity be accompanied by recognizable structural alterations in the tissues. It does not seem valid, therefore, to demand structural change as proof that the tissues are tender to pressure.

### "SITES OF ELECTION" FOR MYALGIC LESIONS

The causes of the myalgic lesion are numerous, though the mechanism by which it is produced is unknown. It may result from trauma (Good, 1942), from established rheumatic disease (Good, 1941), or it may arise *de novo*. Copeman (1943) noticed that tender spots often appeared in the deep tissues after such febrile diseases as influenza; they remained latent, however, until their activity was aroused by further damage in the shape of an injury, a chill or another febrile illness. That these lesions must have some anatomical significance is suggested by their predilection for certain localities. A myalgic spot may occur in muscular tissue anywhere, but the available evidence suggests that they have a strong affinity for musculo-tendinous junctions. The lesions are found most commonly in the erector spinae in the lumbar region, where muscular and tendinous tissues are so interspersed that it is difficult or impossible to tell by palpation exactly the kind of tissue which is involved. The same remark applies to the intercostal muscles. When lesions occur in the larger muscles of the limbs, however, they are found most commonly at a short distance from the attachment of the muscle, where fleshy fibres and tendon should meet. Thus the commonest site for myalgic spots in the buttock is the gluteus maximus near its attachment to the posterior superior spine or the iliac crest (Kellgren, 1938*a*, *b*; Steindler, 1940; etc.).

Pain in the forearm and hand can frequently be attributed to lesions in the extensor group of muscles just below the external epicondyle (Good, 1940; Kelly, 1944*b*), while lesions in the deltoid muscle nearly always occur near its upper or lower attachment. Pain referred to the shoulder, however, is more often caused by lesions in the region of the supraspinous and infraspinous fossae, in which the trapezius and the spinati are most commonly involved (Kelly, 1942; Travell, Rinzler, and Herman).

When pain in the leg is due to myalgic spots, these often can be found in the region of the knee on the inner side, either just above the internal condyle of the femur, or immediately behind the internal condyle of the tibia (Good, 1942; Kelly, 1944*a*). In most cases of painful stiffness of the knee of myalgic origin, the injection of procaine into such lesions will completely remove the disability for a few hours, and in an appreciable number the relief is lasting.

After observing many hundreds of cases of fibrositis, the writer has become convinced that these "sites of election" have some significance in the pathology of fibrositis; so frequently do they become involved that it appears as though the

distribution of deep pain is governed by laws hitherto unknown. It can be shown further that the "sites of election" are tender to pressure in the normal subject. Muscles as a rule display greater sensitivity near their musculo-tendinous junctions than over their bellies; anyone can prove the truth of this statement within a very few minutes. Thus it can easily be demonstrated that the following "sites of election" (which are responsible for about 50% of cases of myalgic pain in the head and limbs) are more tender to pressure than the surrounding muscular tissues: (1) neck muscles just below superior nuchal line; (2) trapezius muscle half-way between acromion and neck; (3) extensor muscles in front of the neck of the radius; (4) gluteus maximus in situation described earlier; (5) deep tissues just above internal femoral condyle.

Case 1 is typical of many in which widely distributed tenderness disappeared upon treatment of a myalgic lesion.

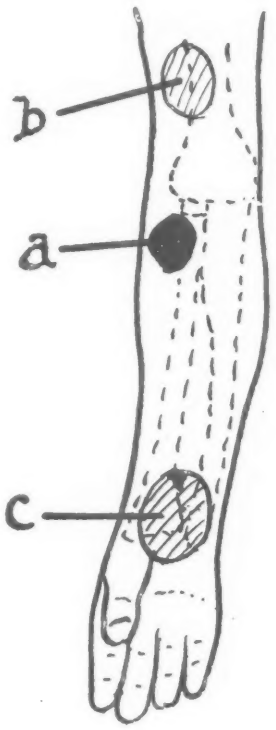


FIG. 1.—Case 1. *a*—Lesion in exterior muscles. *b*—Tenderness related to *Brachialis anterior*. *c*—Tenderness in front of wrist.

J. J. B. suffered from pain in the right shoulder for five months, brought on by exposure to cold in Central Australia. After two months the pain began to radiate to the hand; it seemed to spread from the middle of the humerus to the wrist, with numbness in the thumb and tingling in the index and ring-fingers. No alteration of cutaneous sensation was detected, but three main areas of deep tenderness were found: (1) an oval area, 3″ long, above the external epicondyle in relation to the brachialis anterior muscle; (2) a circular area, 2″ in diameter, in the extensor muscles 3″ below the epicondyle; (3) a circular area, 3″ in diameter, on the front of the wrist. Upon the injection of a few c.c.s. of procaine solution into area (2), the tenderness of the other two areas disappeared as though by magic. Though he had suffered considerably for five months, after the injection he remained completely free from pain. (See Fig. 1.)

### Pressure—Pain

Whatever value attaches to such shadowy observations as these rests upon the recognition of painful pressure, and especially of the intensification of painful pressure, or deep hyperalgesia. Therefore it becomes necessary at this stage to review the present knowledge of the anatomy and physiology of pressure-pain. It can be demonstrated that deep tenderness consists of an intensification of the well-known pressure-discomfort, which normally increases to pain when pressure rises to the required intensity. A certain degree of pressure with the thumb on subcutaneous periosteum, joint-capsule, or tendon will cause a diffuse pain which is not related to any cutaneous sensation, for it can be produced by applying the same pressure through anaesthetized skin. If the pressure is made over a muscle-belly, the pain is more intense and radiates more widely. Certain spots in the muscle, as already described, are more acutely tender. It is possible, by the use of selective local anaesthesia, to demonstrate that the receptors subserving the sensation of pressure-pain reside in the perimysium in the case of muscles, in the peritenal tissues in the case of tendon, in the periosteum in the case of bone, and so on. That the skin and subcutaneous tissues play no part can be demonstrated with ease; thus the pressure required to elicit pain remains the same whether the skin be anaesthetized or no. Where the skin is loose, large folds of skin and subcutaneous tissues can be picked up between finger and thumb and firmly squeezed. If the skin is not sharply deformed by pinching, the greatest possible pressure can be exerted without pain. If muscle is included, however, the well-known sickening sensation of pain is brought on by comparatively light pressure, while the pressure required to elicit pain from tendon is somewhat greater.

#### PREVIOUS RESEARCH ON PAINFUL PRESSURE

Pressure-pain, therefore, is purely a function of the deep tissues. It now remains to localize it more accurately. Judging from the results of recent workers, we should normally expect to find that the pain is elicited from the tissues covering them rather than from the substance of the muscles and tendons. Lewis (1938) and Kellgren (1939) found that pain was not normally elicited from muscle-substance by puncture with a needle, whereas puncture of the deep fascia covering the muscle usually gave rise to the familiar sensation of deep pain. In similar experiments upon tendon, Weddell and Harpman (1940) found that pain was elicited on puncturing the peritenal tissues but not by transfixing the tendon itself. An exception to this was

found in the region of musculo-tendinous junctions, where pain was elicited from the tissue substance; this may be of significance in view of the previously mentioned high sensitivity of such regions to pressure. Weddell and Harpman were able to demonstrate in the peritenon a fine plexus of nerve-fibres, similar to that described in the skin and other organs by Woollard, Weddell, and Harpman (1940). This was regarded as the pain-plexus; it was found in all tissues which gave pain upon pure mechanical stimuli, such as puncture with a fine needle; it could not be demonstrated in muscular tissue, though it was found in the intermuscular fibrous layers and in the perimysium. Thus there has been a happy correspondence between the labours of a number of observers, approaching the subject from different aspects. The present writer, however, could find only a few references to research upon pressure-pain, and these were directed towards its pathways of conduction rather than the tissues in which its receptors are located (Head and Sherren, 1905; Stopford, 1923; Duthie, 1926).

### Experimental Observations

The following experiment was performed a number of times upon himself by the writer. A fine hypodermic needle was passed down to the surface of the muscle (or tendon). The familiar sickening pain indicated that the objective had been reached; the needle then was withdrawn $\frac{1}{4}''$ and 2 c.c.s. of local anaesthetic solution rapidly injected in contact with the surface of the muscle. The needle was withdrawn immediately, and pressure was made at once upon the anaesthetized spot. In every case an area of muscle about 2″ in diameter was found to be completely painless to any pressure which could be applied with the thumb; similarly, when a muscle was pinched between finger and thumb. A small area of deep fascia was anaesthetized on each side of the biceps brachii, or the lower fibres of the gastrocnemius; so long as the tips of finger and thumb were not moved from the anaesthetized deep fascia, no pain was felt. The same results were obtained by compressing the tendo calcaneus after both sides had been anaesthetized.

In every case the skin was normally sensitive, and in every case the pressure was applied so soon after the injection of the procaine that the anaesthetic could not have diffused into the muscle or the tendon. A further experiment was performed upon one of those especially tender spots which are found in the region of the musculo-tendinous junctions. The spot selected was a well-defined one in the inner head of the left gastrocnemius, behind the internal tibial condyle. When the needle reached the sensitive muscle-covering, a peculiarly unpleasant pain was felt, accompanied by twitching of muscle fasciculi. The injection of procaine caused the abolition of pressure-pain; this region was quite as " dead " to pressure as any other area of muscle similarly treated.

#### DEEP HYPERALGESIA

Though these results do but corroborate the observations of earlier workers, they are conclusive as relating to the sense of painful pressure which can be elicited normally from muscle and tendon. It remains now to decide the nature and location of deep tenderness; is this merely an intensification, of normal pressure-pain, residing in the perimysium, or is it more generalized, with normally insensitive muscle-tissue becoming sensitive to pressure? The following experience suggests that it consists of an accentuation of the physiological sensation.

A severe and painful reaction followed the anaesthetization of the sensitive spot in the head of the gastrocnemius. The site of the injection was painful, the pain radiating down to the ankle and halfway up the thigh. The calf muscles were tender over their whole extent, though the site of the lesion was excessively so. No alteration of cutaneous sensation could be detected. A spot was selected, in the centre of the calf, into which 2 c.c.s. of procaine solution were injected just superficial to the surface of the muscle. Within 5 seconds an area of tissue 3″ in diameter was totally insensitive to pressure with the thumb, though no change had taken place in cutaneous sensation (see Fig. 2).

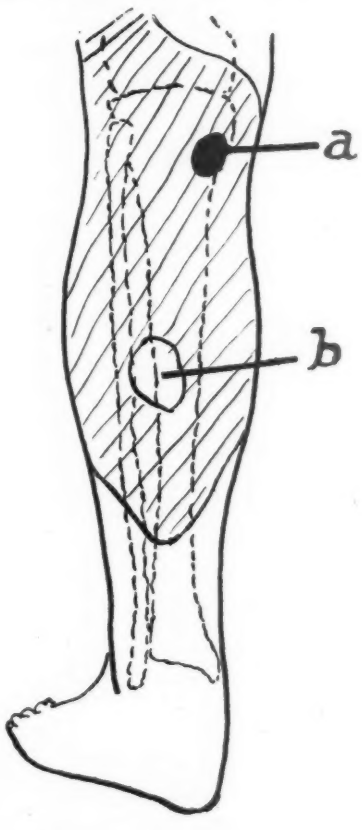


FIG. 2.—*a*—Artificial lesion in inner head of gastrocnemius. Oblique hatching=area of secondary tenderness. *b*—Area in which tenderness was abolished by procaine.

Many similar observations have been made upon the secondary tenderness in patients with fibrositis; in the great majority a small area of tenderness can

be abolished by injecting 1–2 c.c.s. of procaine solution in contact with the surface of the muscle. Pathologically intensified pressure-pain, therefore, consists of increased sensitivity of the fascia overlying the muscle, rather than of abnormal pressure-sensitivity of the muscle itself. This layer contains a plexus of nerve-fibres, with free nerve-endings which are generally acknowledged to be the peripheral receptors of deep pain sensation.

The reflex hypothesis of " referred " deep hyperalgesia is thus supported upon both anatomical and physiological grounds. The picture is singularly complete, and the converging evidence from a number of separate sources cannot be disregarded.

### Fibrositis and the Deep Pain System

If the foregoing remarks are true, the question of the nature of fibrositis must resolve itself largely into an inquiry into the anatomy and physiology of the deep pain system. Nor is this surprising, for the majority of patients with fibrositis have pain and nothing else. Few objective signs are available, and often the only available evidence presented is the assertion of the patient that he feels the pain. Therefore it would seem a more rational procedure to assemble all the available facts regarding pain, than to cling to outworn and unproven conceptions of the nature of fibrositis. There is in fact very little evidence in favour of the generally accepted hypothesis which relates fibrositis to disorders of muscular metabolism. According to this theory, the resultant circulatory disorders lead to lymphatic blockage and to consequent indurations, which cause pain by pressure on nerve-fibres. It was well pointed out by Buckley (1940) that this hypothesis corresponds ill with our present knowledge of sensory function. A critical examination of present views upon fibrositis, says Buckley, is long overdue. The deep pain system, according to Lewis and Kellgren (1939), is anatomically and functionally distinct from the cutaneous pain system. Reflex effects may spread widely in the deep system without affecting the superficial component; and, in those cases in which hyperalgesia of the skin accompanies deep tenderness, there is between the two no correspondence, either in distribution or in time-relationships.

An appreciation of the reality and the importance of the deep pain system will help to throw a good deal of light upon many of the problems of somatic pain. The theory of the reflex spread of hyperalgesia was foreshadowed more than 40 years ago by Gowers (1904), who said that the pain of fibrositis was due to " induced excessive sensibility." In opposition to the reflex hypothesis, it may be argued that, though a nervous mechanism may be sufficient to explain such sensory effects as hyperalgesia, it will not adequately account for the physical changes which may be encountered. The fibroblastic hyperplasia, it may be said, with its surrounding tissue reaction and its later fibrosis, demands more tangible causes of a physical or a chemical nature; it is hard at first glance to believe that palpable indurations in the tissues can result from the effects of disordered nerve-impulses. It should be remembered, however, that very little is known of the relations between the somatic tissues and their nerve-supply, for the reason that it occurs to few people even to think of the possibility of such a relationship. The bulk of the evidence, however, indicates that nervous influences may be responsible for profound tissue changes, both indirectly through alterations in the circulation and directly through effects upon the vital functions of the cells. That acute and careful observer, Sir James Mackenzie (1920, 1926), regarded as the most important of his works the discovery of the part played in the production of symptoms by nervous reflexes. But this, the fruit of the labour of his last few years, went unnoticed by the profession and passed into an oblivion which cannot but be temporary.

### Trophic Nervous Effects

When a sensory nerve is cut, the tissues which it had supplied, after displaying swelling for a few days (Sharpey-Schafer, 1927), become atrophied. When a motor nerve is cut, the corresponding motor fibres atrophy. In like manner, denervated fatty and fibrous tissues undergo retrogressive alterations in structure. Oedema is a recognized effect of interstitial neuritis (Jones and Lovett, 1923; etc.); and the general acceptance of the view that pathological nervous impulses may be responsible for tissue changes is shown by the common use of the term " neuro-pathic oedema." That trophic effects are not wholly due to circulatory changes is shown by the fact that they are not seen after sympathectomy. From this point of view, what appears to be a more important circulatory effect is the vasodilatation which results from the antidromic stimulation of sensory nerves.

### ANTIDROMIC NERVOUS IMPULSES AND AXON REFLEXES

It appears to be no coincidence that both hyperaemia and hyperalgesia of the skin result from impulses propagated antidromically through sensory nerves (Lewis, 1937). In like manner, the hyperalgesia which surrounds a small crushing injury is accompanied, in suitable subjects, by whealing and hyperaemia, both, according to Lewis, the effects of axon reflexes. The fibres which produce hyperaemia appear to be identical with those which produce hyperalgesia; and in both cases they belong to the posterior root system. Lewis thinks it possible that a hitherto unrecognized system of fibres is involved, but Woollard, Weddell, and Harpman pointed out that the pain nerves possessed all the qualities necessary to produce the observed effects. The weight of evidence, in fact, seems to indicate that the pain fibres are responsible (Walshe, 1942).

An analogy was drawn earlier between the superficial and the deep pain fibres; and the fact that cutaneous hyperalgesia is known to spread by nervous reflexes was used to support the proposal

that deep hyperalgesia spreads in an analogous fashion. In like manner, the knowledge that the pain nervous system mediates the spread of hyperaemia in the skin lends great support to the supposition that the deep pain system could be responsible for the propagation of vasodilatation in the deeper tissues. Here again the picture is singularly complete, and the converging evidence once more focuses upon the important point—the implication of the pain nervous system in the production of oedema and tenderness in the deep tissues.

### BI-DIRECTIONAL FUNCTIONS OF SENSORY NERVES

The concept of spread by axon-reflexes involves an idea which is not generally familiar, but which is now well established—the bi-directional propagation of nerve-impulses. The likelihood of bi-directional function in sensory fibres was mentioned by many earlier writers, including Weir Mitchell (1872), Charcot (1877) and Bayliss (1900). The hyperaemia which had been observed to follow antidromic propagation of nerve-impulses was thought by many to be merely a freak effect, with no physiological counterpart. Barron and Matthews (1935*a*, *b*), however, have shown that the branching of sensory axons is much more profuse than had been thought. In the cat and the frog the proximally-directed axon from the root ganglion cell gives off collaterals in the cord, which leave by adjacent posterior roots. In all branches of both the proximal and the distal axons, impulses may run in either direction, so that physiological sensory impulses from one region will give rise to impulses antidromically to sense-organs in another region. The simple mechanism of an axon-reflex, therefore, may be sufficient to explain the reference of pain to regions at a medium distance, but, where a leap of several segments is made, one or more relays must take place in the cord.

### ANALOGY PROVIDED BY SOME PAINFUL POST-TRAUMATIC STATES

An interesting example of a similar mechanism is provided by those painful atrophic conditions which sometimes follow injury to an extremity. Sudeck's atrophy, post-traumatic painful osteoporosis (Miller and de Takats, 1942), minor causalgia (Homans, 1943), post-traumatic spreading neuralgia (Leriche, 1939) and post-traumatic pain syndromes (Livingston, 1943) are terms which have been used by various writers to describe a process which is essentially the same in all instances. Several groups of workers in America have brought forward a good deal of evidence to suggest that the pain, dysaesthesiae, oedema and generalized tissue atrophy are reflex effects, the pathological impulses originating in the damaged tissues. " It seems probable," says Livingston, " that the injury to certain tissues starts a cumulative process which is not confined to a single nerve-distribution, and which tends to spread to involve the spinal cord in diffuse reflex phenomena."

### Physiology of Musculo-Tendinous Junction

If the hypothesis is accepted that most of the manifestations of fibrositis represent only secondary or reflex effects of a myalgic lesion, it must follow that the functional pathology of the lesion differs fundamentally from that of the bulk of the affected tissues. The lesion may arise anywhere in muscular tissues (and perhaps in fibrous and synovial tissues), but it manifests a predilection for some well-defined situations near the junctions of certain muscles with their tendons. Perhaps some significance can be attached to the observation of Weddell and Harpman that pain could be elicited from the substance of the musculo-tendinous junctions, though not from pure tendinous tissue. It can be no mere coincidence, either, that the special neuro-tendinous receptors, or tendon spindles, are found in no other situation than at the musculo-tendinous junctions (Maximow and Bloom, 1934). In each spindle Weddell and Harpman demonstrated in addition to its special sensory nerve-supply an " accessory " nerve-fibre which divides to form in the capsule of the spindle a plexus of unmyelinated fibres, with free nerve-endings. This plexus is indistinguishable from the pain plexus, and the authors say that the naked receptors are the source of the pain which is felt when the tendon receptor is over-stimulated by stretching the muscle.

In the region of the musculo-tendinous junctions, too, are found the majority of the muscle-spindles. In view of the large concentration of sensory organs in its vicinity, there is little reason for wonder that the junction is normally more tender to pressure than the adjacent muscle fibres; nor is it surprising that painful myalgic lesions are found commonly in its neighbourhood. Considering, too, that the muscle spindles and the tendon spindles are the receptor organs for muscular stretch reflexes, it is not surprising that muscular spasm and stiffness usually result from myalgic lesions.

Many interesting questions here suggest themselves, relating to the formation of myalgic lesions in regions apart from the musculo-tendinous junctions; to the formation of multiple lesions simultaneously or in succession; to the causation of the myalgic lesion itself; and to the radiation of the pain in well-recognized patterns. Some speculations upon these points will be reserved for a future article.

### Summary

1. Recent work upon the pathology of fibrositis has added little to the principles laid down by Stockman. Frequently the painful tissues do not present any recognizable histological change.

2. The work of Lewis and his associates upon artificial deep lesions indicates that widespread pain and secondary tenderness may radiate from a localized lesion through the medium of nervous reflexes.

3. The essence of the fibrositic syndrome consists of a localized myalgic lesion together with its more widespread reflex effects.

4. The myalgic lesion shows a predilection for certain localities, mostly in the region of musculotendinous junctions, where concentrations of sensory organs are known to reside.

5. Present knowledge of the deep pain system is reviewed, together with experimental observations intended to locate the site of "pressure-pain" receptors.

6. The reflex theory of fibrositis is shown to be consistent with present knowledge of deep sensory function, in which axon-reflexes and antidromic impulses play a large part.

The Director-General of Medical Services, Australian Military Forces, has kindly permitted the publication of this essay.

*Addendum.*—Since this article was set into type I have had the opportunity of reading in full the excellent contribution by Copeman and Ackerman. The correlation of the "fat pattern" of the back offers a fascinating field for study. Also, perusal of further work by Barron (*J. Neurophysiol*, 1940, **3**, 403) shows that the original findings of Barron and Matthews are not confirmed in all particulars. It now seems unlikely that collaterals leave the cord by adjacent roots; but the reflex spread of sensory impulses, through profuse branchings, has been amply confirmed.

## References


Abel, O., Siebert, W. J., and Earp, R. (1939). *J. Mo. med. Ass.*, **36**, 435.
Barron, D. H., and Matthews, B. H. C. (1935a). *J. Physiol.*, **85**, 73.
——, ——, (1935b). *Ibid.*, **85**, 104.
Bayliss, W. M. (1900). *Ibid.*, **26**, 173.
Buckley, C. W. (1940). *Ann. Rheum. Dis.*, **2**, 83.
Charcot, J. M. (1877). *Lectures on Diseases of the Nervous System*. London.
Collins, D. H. (1940). *Ann. rheum. Dis.*, **2**, 114.
Copeman, W. S. C. (1943). *Ibid.*, **3**, 222.
—— and Ackerman, W. L. (1944). *Quart. J. Med.*, **3**, **13**, 37. (Cited in *Med. J. Austral.*, (1945), **1**, 229.)
Duthie, O. M. (1926). *Brain*, **49**, 187.
Ganado, W. (1945). *Brit. med. J.*, **1**, 141.
Good, M. G. (1940). *Lancet*, **2**, 326.
—— (1941). *Practitioner*, **146**, 167.
—— (1942). *Ibid.*, **148**, 236.
Gowers, W. R. (1904). *Brit. med. J.*, **1**, 117.
Head, H., and Sherren, J. (1905). *Brain*, **28**, 116.
Hench, P. S., Bauer, W., Dawson, M. H., Hall, F., Holbrook, W. P., Key, J. A., and McEwen, C. (1941). *Ann. intern. Med.*, **14**, 1383.
——, ——, Boland, W., Dawson, M. H., Freyberg, R. H., Holbrook, W. P., Key, J. A., Lockie, L. M., and McEwen, C. (1941). *Ibid.*, **15**, 1002.
Homans, J. (1943). *Amer. J. med. Sci.*, **205**, 313.
Inman, V. T., and Saunders, J. B. de C. M. (1944). *J. nerv. ment. Dis.*, **99**, 660.
Jones, R., and Lovett, R. W. (1923). "Orthopaedic Surgery." London.
Jordan, H. H. (1941). *N.Y. St. J. Med.*, **41**, 2203. (Cited by Travell *et al.*)
Kellgren, J. H. (1938a). *Brit. med. J.*, **1**, 325.
—— (1938b). *Clin. Sci.*, **3**, 175.
—— (1939). *Ibid.*, **4**, 35.
Kelly, M. (1942). *Med. J. Austral.*, **1**, 311.
—— (1944a). *Ibid.*, **1**, 286.
—— (1944b). *Ibid.*, **2**, 185.
Leriche, R. (1939). "The Surgery of Pain." London.
Lewis, T. (1937). *Brit. med. J.*, **1**, 431, 491.
—— (1938). *Ibid.*, **1**, 321.
—— (1942). "Pain." New York.
—— and Kellgren, J. H. (1939). *Clin. Sci.*, **4**, 47.
Livingston, W. K. (1943). "Pain Mechanisms." New York.
Mackenzie, J. (1920). "Symptoms and Their Interpretation" (Appendix). London.
—— (1926). "The Basis of Vital Activity." London.
Maximow, A. A., and Bloom, W. (1934). "A Text-Book of Histology." Philadelphia.
Miller, D. S., and de Takats, G. (1942). *Surg. Gynec Obstet.*, **75**, 558.
Mitchell, S. W. (1872). "Injuries of Nerves and Their Consequences." Philadelphia.
Sharpey-Schafer, E. A. (1927). *Brain*, **50**, 538.
Steindler, A. (1940). *J. Bone Jt. Surg.*, **22**, 28.
Stockman, R. (1920). "Rheumatism and Arthritis." Edinburgh.
Stopford, J. S. B. (1923). *J. Anat.*, Lond., **57**, 199.
Travell, J., Rinzler, S., and Herman, M. (1942). *J. Amer. med. Ass.*, **120**, 417.
Walshe, F. M. R. (1942). *Brain*, **65**, 48.
Weddell, G., and Harpman, J. A. (1940). *J. Neurol. Psychiat.*, n.s., **3**, 319.
Woollard, H. H., Weddell, G., and Harpman, J. A. (1940). *J. Anat. Lond.*, **74**, 413.

# A STUDY OF RHEUMATISM IN A GROUP OF SOLDIERS WITH REFERENCE TO THE INCIDENCE OF TRIGGER POINTS AND FIBROSITIC NODULES

BY

L. G. C. E. PUGH and T. A. CHRISTIE

The nature and significance of the trigger points and nodules characteristically present in patients suffering from acute attacks of fibrositis has been discussed in recent papers by Copeman and Elliott. Elliott, using the myoscillagraph, has shown that areas of localized tenderness on deep palpation may be due to subclinical spasm in portions of the underlying muscle, and suggests various ways in which this state of spasm may arise. Copeman on the other hand believes that the tender nodules and trigger points are the clinical signs of changes in fibro-fatty tissues involving oedema and in some cases herniation of discrete lobules of fat.

We have approached the subject from a different angle by examining a group of healthy soldiers for the presence of nodules, tender or otherwise, and of trigger points. We also obtained from each individual information as to whether he had at any time in his life suffered from pains in the joints, pain and stiffness of the back, or other symptoms of the rheumatic group of diseases. After excluding histories readily explained on non-rheumatic grounds, the results were compared with the incidence and distribution of trigger points and nodules which had been found. Owing to the lack of precision of a diagnosis founded on past history, it seemed wiser to choose a generic term of description for the positive cases rather than seek to distinguish between different types; and we have, therefore, employed the term " rheumatism," and have referred to individuals giving positive histories as " rheumatic subjects."

Five hundred and twenty-two members of a territorial regiment, R.A. were examined in Holland in the spring of 1945. The majority had served with the unit since the outbreak of war, for which reason their ages showed a somewhat unusual distribution for a forward unit having a range of 19 to 42, with modes of 25 and 33 years. The men had been living in tents on low-lying water-logged ground in the Rhine delta. They had been in action continuously all the winter. In spite of the cold and damp living conditions and the strain of battle, the health of the troops had been good. We were particularly impressed by the low sick rate due to rheumatism and attributed this to the quality of the personnel and their high morale. The fact that the men were thoroughly conditioned to exposure was probably also an important factor contributing to the low incidence of rheumatic disability.

## Method

The back, shoulders, and buttocks of each individual was examined for the presence of trigger points and palpable nodules. A trigger point is defined as a circumscribed area of tenderness, firm palpation of which causes pain and reflex contraction of the adjacent or underlying muscles. Upon the conclusion of this examination the rheumatic history was taken. The following symptoms, unless adequately explained by anticedent trauma, postural defects, unaccustomed exertion, or an incipient febrile illness, were accepted as falling within the rheumatic category: stiffness and pain of muscles and joints during movement; pain and weakness on violent movement; occasional and transitory stabbing pain on initiating movements; periodic aching pain referred to joints or muscles and present during rest.

## Results

The results of this investigation are set out in Tables I and II.

TABLE I.—SHOWING RELATIONSHIP BETWEEN THE RHEUMATIC DISPOSITION AND THE PRESENCE OF TRIGGER POINTS

| | Number of Individuals | | Total |
|---|---|---|---|
| | Trigger points present | Trigger points absent | |
| Rheumatic subjects .. | 46 (32%) | 97 (68%) | 143 |
| Non-rheumatic subjects .. .. | 11 (3%) | 368 (97%) | 379 |
| Total .. | 57 (11%) | 465 (87%) | 522 |

*Note.*—Five cases of rheumatic fever in adolescence without subsequent symptoms, and one case of rheumatic fever in January, 1945, are included in Table I but omitted from Table II.

TABLE II.—CLASSIFICATION OF RHEUMATIC CASES ACCORDING TO PART OF BODY PRINCIPALLY AFFECTED, AND FREQUENCY OF TRIGGER POINTS IN EACH GROUP

| | Trigger points present | Trigger points absent | Total | |
|---|---|---|---|---|
| Back only .. .. | 25 | 29 | 54 | 61 |
| Back and other joints | 4 | 3 | 7 | |
| Shoulders only .. | 9 | 31 | 40 | 47 |
| Shoulders and other joints .. .. | 2 | 5 | 7 | |
| Hips only .. .. | 1 | 6 | 7 | 10 |
| Hips and other joints | 1 | 2 | 3 | |
| Knees only .. .. | — | 4 | 4 | 10 |
| Knees and other joints | 2 | 4 | 6 | |
| Muscles of thighs and legs .. .. | — | 6 | 6 | 6 |
| Ankles only .. .. | — | 1 | 1 | 1 |
| Wrists and elbows .. | 1 | 1 | 2 | 2 |
| Total .. | 45 | 92 | 137 | |

### Rheumatic History

In 143 out of 522 individuals (28%) a positive history of rheumatic symptoms was obtained. In the majority of cases these symptoms were of a minor and transient character, not requiring treatment. They were associated particularly with changes of weather, periods of cold and damp, the wearing of wet clothes, or sleeping between damp blankets. Of those more severely affected not all had reported sick; and there were some who in spite of chronic pain and stiffness carried on with their work for fear of having to leave the unit. Ninety-three out of 143 subjects attributed their symptoms directly to climatic influences, and only 10 had been free of symptoms during the previous winter. At the time of this investigation the weather had been warm and dry for the past three weeks and there were only six then complaining of rheumatic pain.

The classification of cases given in Table II shows that the back or upper part of the body was affected predominantly, in greater frequency than the lower limb; the proportions being: back 45%, shoulders and upper limb 35%, lower limb 20%. From previous experience, it is considered probable that this distribution is related to the nature of the work in a heavy artillery regiment, involving as it does strenuous lifting, heaving, and pushing, and that a similar investigation carried out on an infantry unit would reveal a relatively higher incidence of involvement of the lower extremity.

Of 14 men who had suffered from rheumatic fever in adolescence, 9 complained of occasional pain and stiffness in the joints brought on by exposure, and affecting principally the knees and shoulders (in 2 cases the back also); the other 5 had remained free of symptoms since the original attack. One individual was encountered who had his first attack of rheumatic fever three months previously at the age of 28 years.

### Nodules

Palpable nodules were found to be present in 16% of rheumatic and 15% of non-rheumatic individuals; hence it is concluded that as far as rheumatism is concerned their presence is of no significance. They occurred with equal frequency in the region below the iliac crests and along the borders of the scapulae, but were not found in the lumbar region of the back, a common site for trigger points. When nodules were discovered in rheumatic subjects, in half the cases one or more of them was tender, and the tender ones were always located in the iliac region. The finding of tender nodules below the iliac crests has led Elliott (1944) to suggest that the quality of tenderness in nodules depends merely on their situation in an area where on palpation they are compressed against bone or ligament. In the present investigation, however, tender as opposed to non-tender nodules were present four times as often in rheumatic as in non-rheumatic subjects. The quality of tenderness therefore appears definitely to be associated with a history of rheumatic pain.

### Trigger Points

Trigger points were found in 30% of rheumatic and 3% of apparently non-rheumatic subjects. The association of trigger points with a history of rheumatic symptoms is therefore a strong one. Tender nodules are included in this result since it appears to be the quality of tenderness that is significant and not palpability. They therefore come within the definition of trigger points and are included under that heading in Tables I and II. The situations in which trigger points were located were as follows: the lateral borders of the paravertebral muscles; the sacral region; below the iliac crests; medial to the vertebral border of the scapula; the upper border of the trapezius muscle; in the supra-scapular region. It may be noted that most of these situations conform to the areas of the basic fat pads defined by Copeman (1944) as being sites in which trigger points are most commonly to be found in the acute stage of attacks of fibrositis.

In the rheumatic subjects trigger points were situated in regions previously affected by symptoms: e.g. along the lateral borders of the paravertebral muscles in the cases of lumbago; or in areas from which pain commonly radiates in the acute stage, e.g. the upper border of trapezius or the scapula region in the case of fibrositis of the upper part of the back and shoulders. Out of 45 subjects there were only 3 in whom the site of the trigger points was unrelated to the region affected by the rheumatic symptoms.

### Discussion

The symptoms described by most of the rheumatic subjects were characteristic of fibrositis. In view of the high incidence in a fit body of men of high morale and the close relationship to climatic factors, it appears to us unlikely that such symptoms

can be regarded as entirely psychogenic. On the other hand, since men of low morale and unstable personality tend to exaggerate and prolong their minor disabilities, there is no doubt that among rheumatic subjects reporting sick and receiving medical treatment a high proportion show neurotic traits. So common is this finding that several authorities, in the absence of unequivocal pathological information, have been led to doubt the existence of a physical basis for fibrositis.

Similarly, the persistence of trigger points in the absence of symptoms in 30% of rheumatic compared with 3% of non-rheumatic subjects might be explained in terms of " function," both symptoms and trigger points being interpreted as signifying a low threshold for sensations of pain. But since the trigger points, though conforming to a general pattern, were seldom symmetrical in distribution and often occurred singly, we submit that they are more readily interpreted as evidence of some persisting structural or biochemical change in the tissues, the nature of which will eventually be determined. Copeman (1944, 1945) has adduced evidence that trigger points are the result of pathological changes in fibro-fatty tissues, and not in muscle or fibrous tissue. Elliott (1944), on the other hand, has recently suggested that in some cases of rheumatic pain, trigger points are due to circumscribed areas of muscle spasm. The results of the present investigation lend support to the former view, for it is difficult to accept the possibility of muscle spasm persisting in the absence of symptoms. In the acute stage of an attack of fibrositis, on the other hand, muscle spasm may well arise as a result of reflex irritation from the specific lesions in neighbouring tissues. Such a mechanism has clearly been demonstrated in the case of sciatica due to prolapse of an intervertebral disc, and is held to be responsible in part for the pain in this condition. Similarly in fibrositis, the symptoms may have their origin partly in the fibrositic lesion, and partly in the associated muscle spasm.

## Summary

1. Of 512 fit soldiers, 146 (28%) gave a previous history of pain and stiffness of muscles and joints, considered to be of rheumatic origin.

2. Of these 146 rheumatic subjects, trigger points or tender nodules were found to be present on palpation in 46 (32%), although only 6 had symptoms at the time of examination.

3. Among 379 apparently non-rheumatic subjects, tender nodules or trigger points were found in only 11 (3%).

4. Non-tender nodules were found in equal proportions of rheumatic and non-rheumatic subjects, and are therefore considered to have no rheumatic significance.

5. The significance of these results is discussed.

In conclusion we wish to express our gratitude to Lt.-Col. Burrows, R.A., for granting permission for this work to be carried out, and to Lt.-Col. W. S. C. Copeman, R.A.M.C., for his advice and encouragement in the preparation of this paper.

## References

Copeman, W. S. C. and Ackerman, W. L. (1944). *Quart. J. Med.* **13**, 37.

Elliott, F. A. (1944). *Ann. rheum. Dis.*, **4**, 22.

# OBSERVATIONS ON THE TREATMENT OF RHEUMATIC FEVER WITH VITAMIN P

BY

J. F. RINEHART

*University of California Medical School, San Francisco*

Recently, in this journal (1943), the writer reviewed evidence pertaining to the possible rôle of nutritional deficiency as a contributory factor in the aetiology of rheumatic fever. In this report it was shown that a series of experimental, epidemiological and clinical considerations implicated vitamin C deficiency as a factor in the pathogenesis of rheumatic fever. While vitamin C deficiency may be important in predisposing to the initial attack of rheumatic fever and may, *per se*, be of some value in treatment, ascorbic acid has failed to exert any marked influence on the course of the illness or to prevent recurrences.

In 1941 we became interested in the possible therapeutic value of vitamin P in rheumatic fever and, in the paper referred to, reported on the apparent favourable influence of this substance in 3 cases of persistently active rheumatic fever. Subsequently, we recorded preliminary observations on small numbers of cases of rheumatic fever so treated (1944). Vitamin P was so designated because of its reputed influence on capillary permeability. In the excellent original report of Armentano and his colleagues (1936), evidence was presented that this substance was effective in correcting the abnormal capillary fragility in cases of vascular purpura as well as restoring to normal the increased permeability of capillaries encountered in certain infections. It is of interest that most cases of so-called vascular purpura probably have an allergic basis. Considerable evidence suggests the operation of allergic factors in rheumatic fever and the frequent occurrence of haemorrhagic manifestations is well known. The plant pigment or flavone constituting vitamin P is considered to act in conjunction with ascorbic acid. In view of these considerations and the early encouraging observations made in treatment of rheumatic fever it is natural that we should consider it desirable to extend investigations in this field.

It is the purpose of this paper briefly to report observations which we have made on 39 cases of rheumatic fever which have been treated with vitamin P for periods of one month or longer. A high proportion of the cases have been of the polycyclic or refractory type. All showed activity of the rheumatic process at the time treatment was instituted, as evidenced by the sedimentation rate The diagnosis of rheumatic fever has been established beyond reasonable question in all cases. In many an accelerated sedimentation rate was the only clear evidence of continued activity of the disease. Inasmuch as sedimentation rates had been determined by different methods, for purposes of analysis and graphic representation, it was necessary to establish appropriate equivalents for the several methods applied. While we have not been able to establish absolutely accurate comparative scales, they are entirely adequate for the purposes. In most cases a single method was used in individual cases.

## Observations

Analysis of the data reveals the following. Of the 39 cases, 24 were children and 15 were adults. Twenty-six of the 39 cases had exhibited persistent activity for periods of 6 weeks or longer in spite of the application of the usual methods of management. The average duration of the illness in this group was 10 weeks. The average sedimentation rate of the 39 cases at the time treatment was instituted was 33 mm. per hour in terms of the Wintrobe scale. One month from the institution of treatment the average sedimentation rate was 17·5 mm. per hour. Thirty-four of the 39 cases exhibited significant slowing of the sedimentation rate at the end of 6 weeks (4 weeks in 12 of the cases in which observation was terminated at the time). Two cases showed no significant changes in sedimentation rate; in 3 the rate of sedimentation was more rapid. Two of the latter suffered obvious intercurrent infection which would account for this. Between 4 and 6 weeks after therapy 22 of the 39 cases showed either no evidence of activity or minimal activity.

The findings in the 26 persistent cases are of particular interest. In this group the average initial sedimentation rate was 32 mm. per hour and at the end of 1 month was 15·5 mm. per hour (Wintrobe scale). It is noteworthy that this occurred in such a short period of time in a group of cases that, under observation, had shown persistent activity over considerable periods of time. At the end of 1 month, 22 of the 26 cases in this

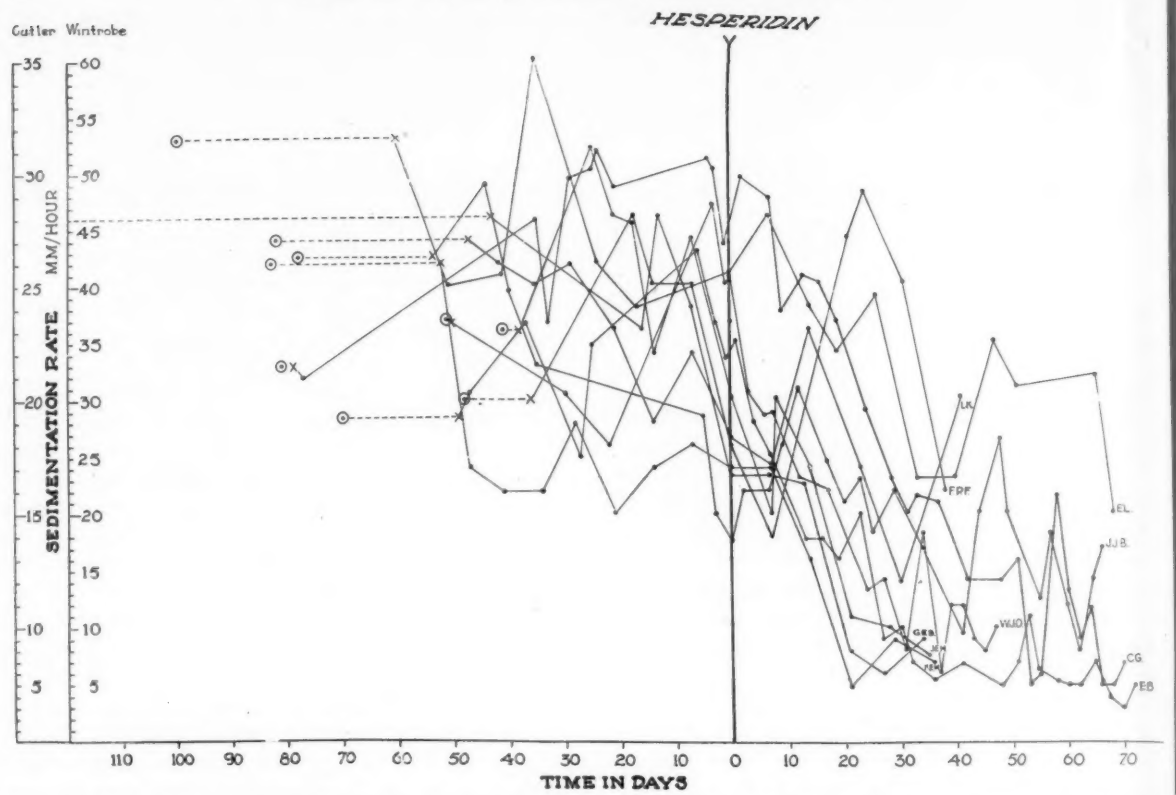


FIG. 1.—Curves of sedimentation rate in 16 cases of persistent rheumatic fever in children treated with hesperidin. The circles indicate time of clinical onset; the crosses indicate the first recorded sedimentation rate and the dots indicate subsequent determination.

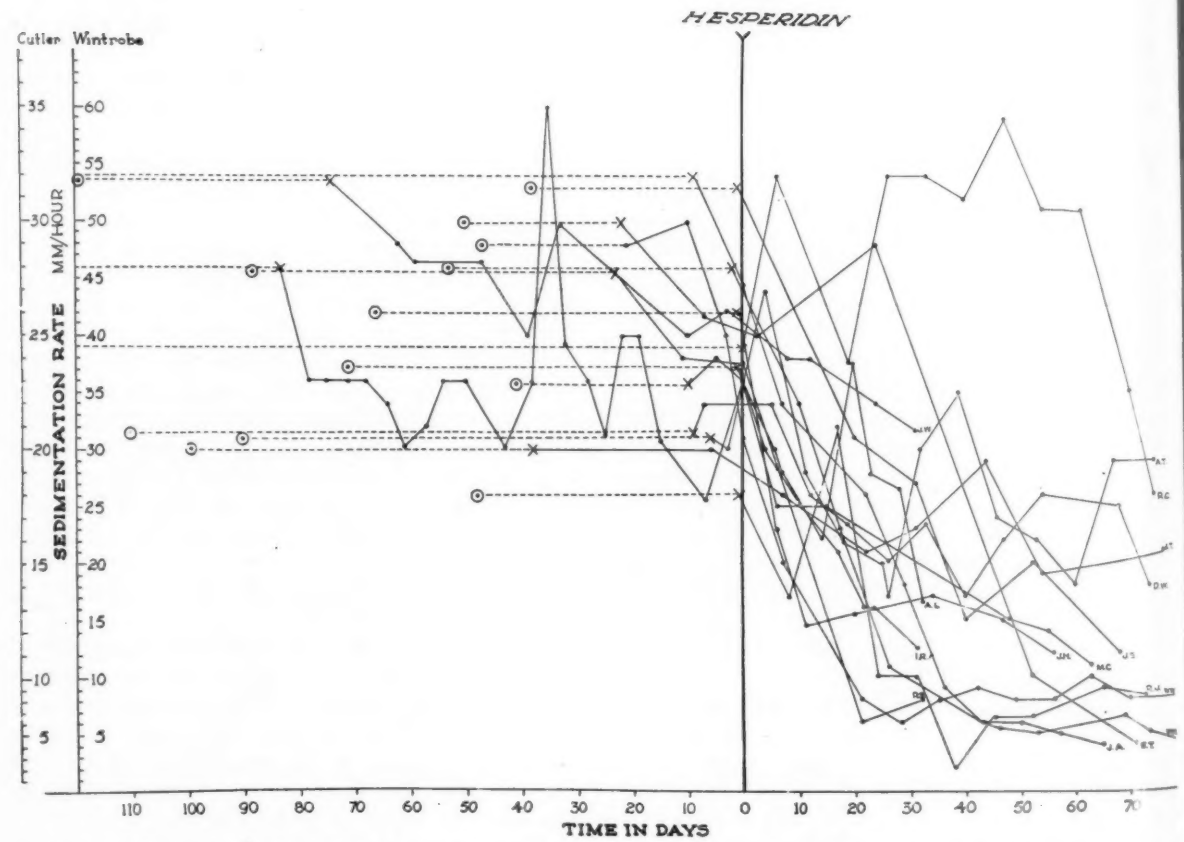


FIG. 2.—Curves of sedimentation rate in 10 adult cases of persistent rheumatic fever treated with hesperidin. Symbols as in Fig. 1.

group had exhibited significant slowing of the sedimentation rate. In 3 the rate was somewhat more rapid and in 1 it was unchanged. Sixteen cases showed either minimal or no activity at the end of one month. The graphic representation of the sedimentation rates shown in Figs. 1 and 2, suggests most strongly that improvement was related to the treatment.

Emphasis has been placed upon the sedimentation rate in this study because it is the most critical objective index of activity and gauge of progress in cases in which the diagnosis of rheumatic fever is established. In many it was the only definite evidence of continued active disease. Insofar as other manifestations of activity existed, improvement occurred which paralleled the slowing sedimentation rate.

## Discussion

It is considered highly probable by the writer that the slowing of the sedimentation rate in the cases reported is related to the treatment. This is believed to be so particularly in the persistent cases where clinical observations indicated prolonged activity prior to treatment. It will be noted by reference to Figs. 1 and 2 that many cases showed significant slowing of the sedimentation rate within 2 weeks. The cases of relatively short duration, although showing similar responses, are less convincing in that spontaneous recovery is frequently observed in a 4 to 6 week period. Although the persistent cases act to some extent as their own control, it is naturally desirable to extend the studies utilizing alternate cases as controls. This is being done at the present time, and the data now available indicates that the treated cases are showing more favourable response than the untreated controls.

It should be pointed out that in the cases studied, vitamin P was given as additional therapy, i.e. it was added to the therapeutic regime of the patient at the time study of the case was undertaken. Although it has not been studied, we have no evidence that the substance possesses analgesic or antipyretic properties analogous to salicylates. The vitamin P-containing material which we have used in most of the cases is crude Hesperidin which has been fortified with Hesperidin Methyl Chalcone.* It is prepared in compressed tablets containing 0·5 grams of crude Hesperidin and 20 mg. of Hesperidin Methyl Chalcone. The usual dosage has been 1·5 grams; one tablet is given three times daily with meals. No toxic or ill effects have been observed which are ascribable to this medication. In a number of cases the material has been administered for several months. The mode of action is uncertain. If vitamin P acts as a co-enzyme with vitamin C as suggested by Szent-Györgyi (1939), deficiency of this factor would block effective utilization of ascorbic acid. Also, it is of interest that the cases of vascular purpura which are reported as benefited by vitamin P are generally considered to be of allergic nature. There is considerable evidence that an allergic mechanism operates in rheumatic fever.

In view of the great importance of rheumatic fever as a cause of cardiac disability and death, studies of promise should be pursued with vigour. If nutritional deficiency is a contributory aetiologic factor, it is evident that prevention would be feasible.

## Summary

Observations are recorded in 39 cases of rheumatic fever treated with vitamin P. Evidence is presented that this substance exerts a beneficial influence on the course of the illness as reflected chiefly by slowing the sedimentation rate. Extension of studies on the application of vitamin P in treatment and prophylaxis of rheumatic fever are indicated.

The writer gratefully acknowledges the cooperation of Dr. Helen Johnson, Chief of the Cardiac Programme of the Crippled Children's Bureau of the California State Department of Public Health.

## References

Armentano, L., Bentsáth, A., Béres, T., Rusznyák, Stefan, and Szent-Györgyi, Albert (1935). *Dtsche med. Wschr.*, 1936, **36**, 1325.
Rinehart, J. F. (1943). *Ann. rheum. Dis.*, **3**, 154.
——, and Johnson Helen (1944). *J. Clin. Invest*, **23**, 941.
—— (1944). *Calif. Hlth.*, **1**, 163.
Szent-Györgyi, Albert von (1939). "On Oxidation, Fermentation, Vitamins, Health and Disease." Baltimore, Maryland.

* We are indebted to The Abbott Laboratories for liberal supplies of the fortified Crude Hesperidin.

# A BENIGN TYPE OF RHEUMATIC FEVER

BY

W. L. ACKERMAN

A diagnosis of rheumatic fever conjures in the minds of most physicians a serious disease demanding prolonged rest in bed, suggesting the likelihood of cardiac damage, and producing subsequent invalidism to a varying degree. Copeman (1944) described 42 cases of febrile polyarthritis of acute onset which he states were " non-epidemic, of short duration, and probably not uncommon." None of his cases developed cardiac damage, and all occurred in patients whose previous resistance to disease appears to have been exceptionally high. For this benign syndrome he suggests the name " acute febrile myalgia " as being better than " benign rheumatic fever " for several reasons, in spite of the prominence of the arthritic manifestations.

Ferguson (1944), in reviewing 243 such cases occurring in the Canadian Army, suggested the appellation " acute febrile polyarthritis " for a condition in adults simulating rheumatic fever, but which has not the gloomy prognosis of this disease as seen in children. The diagnosis of rheumatic disease was usually followed in the Canadian Army by discharge from the Service (162 out of 243). In his opinion at least half of those who developed the condition and were discharged could continue in military service. W. Tegner (1944), the reviewer of Ferguson's article, could not agree that a change in nomenclature is needed. He, however, agreed that a diagnosis of rheumatic fever should not automatically entail discharge from the Army.

The 47 Service patients discussed in this paper suffered with this " benign type " of rheumatic fever and have been seen within the last 2½ years. They showed an acute febrile illness with non-suppurative polyarthritis simulating classical rheumatic fever, yet with features which differentiated them from this serious disease, and enabled a shorter period away from duty to be imposed, and a more optimistic prognosis to be given, which within the limits of a relatively short follow-up proved to be justified. During this period only 7 cases of classical rheumatic fever with endocarditis were seen, and in none of these was the attack the primary one.

## Typical Case Reports

### CASE 1

Aged 28. No previous history of rheumatic fever or rheumatism. No history of tonsillitis—tonsils removed 10 years ago. For past four months had been exposed to severe cold and wet and had not been able to dry his clothes properly. Two days before admission began to complain of pain and stiffness in several joints, particularly L. elbow and R. knee. The following day both knees became obviously swollen.

*On Admission—May* 7 : O.E. Well developed man. T. 102·8°, P. 102. Does not look ill. Slight sweating. No tonsil remnants seen. Marked effusion into both knee joints; periarticular swelling L. ankle. These joints are painful on movement and palpation. Less severe pain R. wrist and R. shoulder. Treated with sod. sal. grs. 120 daily.

*Progress.—May* 9: T. 98·6, P. 74. Pain less, has almost disappeared from L. ankle, R. wrist, and R. shoulder. Knees still painful and swollen. B.S.R. =42 mm. in 1 hour. *May* 11: T. 98·2°, P. 70. Disability limited to both knees which are still moderately swollen and painful. *May* 13: T. normal, P. 70. No swelling of knees. B.S.R.=42. *May* 14: Sod. sal. reduced to 90 grains daily. B.S.R.=40. Slight stiffness in knees. R.B.C.=5,200,000. C.I.=1·0. *May* 20: Feels well; remains apyrexial; slight stiffness in knees. *May* 24: B.S.R.=24. Sod. sal. stopped. *May* 28: B.S.R.=14. Begins getting up for gradually increasing periods. Heart remains unaffected. *June* 16: Up all day. No complaints. B.S.R.=10. *June* 28: Returned to duty. Well.

### CASE 2

Aged 26. For past two weeks had been undergoing severe training during which he had frequently become soaked by rain: " had been living rough." Frequent twinges of pain in limbs and back during this time. For past three days had had difficulty in performing his duties owing to pain in ankles and feet. Several mild attacks of lumbago during his training as a soldier. No history of sore throat or previous rheumatism.

*Dec.* 12: O.E. Well developed man. T. 102°, P. 96. Complains of pain in various joints. Joints affected are: both ankles (some periarticular swelling with pain on movement), both knees (pains and stiffness only), and L. shoulder (restricted movement owing to pain). Given sod. sal. grs. 120 daily. *Dec.* 13: T. 102·6°, P. 100. Pain more severe. Effusion into L. knee joint. B.S.R.=34. *Dec.* 14: T. 102°, P. 96. No change in physical signs. Sod. sal. increased to 150 grs. daily. *Dec.* 15: T. normal, P. 80. Periarticular swelling of ankles had disappeared, some pain still present on movement. Effusion still persists in L. knee. *Dec.* 18: Disability limited to very slight effusion L. knee. B.S.R. =30 mm. R.B.C=4,800,000. C.I.=1·2. Sod. sal. reduced to 90 grs. daily. *Dec.* 24: No symptoms. B.S.R.=24 mm. Sod. sal. stopped. *Dec.* 16: B.S.R. =16 mm. *Jan.* 1: B.S.R.=12. Heart has remained unaffected throughout. *Jan* 24: Uneventful progress. Discharged to duty.

CASE 3

Aged 23. 2 days sore throat. *P.H.* Thinks he had rheumatic fever aged 10. Nil since. In bed 5 weeks. Has been susceptible to sore throats with occasionally a muscular pain in his back; otherwise has always considered himself to be very fit.

*Sept.* 12: O.E. T. 103·4°, P. 100. Bilateral acute follicular tonsillitis with peritonsillar induration. Swab showed hæmolytic streptococci. No other abnormal physical signs on examination. Sulphonilamide 2 grammes stat. and 1 gramme 4-hourly. *Sept.* 14: T. normal. Throat has responded well to sulphonilamide. Peritonsillar induration has disappeared. Slight follicular tonsillitis. *Sept.* 17: T. 102, P. 94. Complains of pain in knees and wrists. Effusion into L. knee. No obvious tonsillitis. Sulphonamide stopped. (36 grammes) Sod. sal. grs. 150 daily begun. *Sept.* 18: T. 102·6°, P. 100. Pain more severe. Effusion L. knee has increased. Periarticular swelling and puffiness L. ankle. c/o severe pains in wrist; L. wrist slightly swollen. B.S.R.=48 mm. *Sept.* 20: T. 99·2°, P. 96. Effusion both knees, less pain. Induration around L. ankle has disappeared. Heart=N.A.D. R.B.C.=5,000,000. C.I. =1·1. Swab from tonsillar region=haemolytic streptococci. *Sept.* 22: T. normal, P. 88. Effusion much less in knees. Slight pains in other joints. *Sept.* 26: T. normal, no effusions. Very slight pain in knees. Sod. sal. reduced to 90 grs. daily. *Sept.* 29: B.S.R.=20. No symptoms or signs. Sod. sal. ceased. *Oct.* 4: B.S.R.=16 mm. *Oct.* 10: B.S.R.=4. Begins to get up. *Oct.* 21: Discharged to light duty. *Oct.* 24: Returns for tonsillectomy: has had no recurrence of symptoms. Heart—N.A.D.

CASE 4

Aged 32. *Aug.* 29: Pain and swelling in both ankles —he thought it due to route marches. Persisted for two days but not sufficiently severe to prevent his embarking in a landing craft. No previous rheumatic or other illness. No undue exposure to cold or damp. Tonsils removed as a child. *Sept.* 3: Recurrence of pain in ankles, also appeared in knees, persisted. *Sept.* 5: O.E. T. 102·8°, P. 98. Marked painful effusions into knees, L. and R. Both ankles swollen and painful. Tenderness and induration L. wrist. Pain on moving L. elbow and shoulder. Tonsils had been radically removed. Sod. sal. grs. 150 daily. *Sept.* 7: T. 102°, P. 90. Knees as before, both wrists now affected. Ankles appear normal except for slight pain on movement. B.S.R.=32 mm. *Sept.* 9: T. normal, P. 74. Slight effusion both knees. Wrists less swollen. *Sept.* 12: Remains apyrexial. P. 68. Subjective symptoms in knees. Cease sod. sal. *Sept.* 18: Feels well. Heart remains clinically unaffected. B.S.R. =32 mm. *Sept.* 28: B.S.R.=20. Allowed up for increasing periods. *Oct.* 3: Sudden recurrence of pain in knees, wrists and L. ankle. *Oct.* 3: T. 101°, P. 80. Mild effusion into knees. B.S.R.=28 mm. Sod. sal. grs. 150 recommenced. *Oct.* 5: T. normal, P. 76. Pain in joints much less. Slight effusion still present in knees. *Oct.* 9: Remains apyrexial. P. 68. No pain. No effusion. Sod. sal. reduced to grs. 90 daily. *Oct.* 14: Sod. sal. ceased. *Oct.* 18: B.S.R.=20 mm. *Oct.* 26: Feels well. B.S.R.=8 mm. Recommenced getting up. *Nov.* 16: Discharged. Heart remains unaffected throughout illness.

SYMPTOMS AND SIGNS

The average age of these 47 male patients was 25·2 years. The onset of the illness was fairly sudden with pyrexia (up to 103·8°). Stiffness and pain occurred in the joints, rapidly followed in many by effusions. The joints involved were chiefly the ankles, knees, wrists and elbows, the hips and shoulders being occasionally implicated. Moderate periarticular swelling was frequently present in the wrists and ankles. There was very little tendency for the pains to flit from joint to joint. There was generally no previous history of rheumatism or other serious illness. The patient rarely felt acutely ill, the discomfort associated with the joints being the outstanding complaint. Profuse sweating was not noticed. Any increase in the pulse rate corresponded with the pyrexia. No rheumatic nodule nor any of the erythemata was seen. In all the patients (except 2), the blood sedimentation rate was raised, generally 30–40 mm. in one hour (Wintrobe). Examination of the red blood cells and haemoglobin in 19 cases showed a normal count.

PROGRESS

All were treated with sodium salicylate 120–150 grs. daily. The clinical improvement was rapid. The swelling of the joints usually disappeared in 5 days although arthritic discomfort was frequently perpetuated for several more days. Almost invariably the patient quickly protested at being confined to bed. All but 2 were kept in bed until the B.S.R. had fallen to normal (average 24 days) after which they were allowed up for gradually increasing periods, as the sedimentation rate is considered by the majority of investigators as the most important simple laboratory test of rheumatic activity. Recurrences and relapses, so common in rheumatic fever, were rare. Three patients relapsed soon after beginning to get up. Two of these had been allowed to exercise before the B.S.R. had fallen to satisfactory levels. The response of these relapses to bed and salicylates was as prompt as in the original attack. None developed endocarditis or myocarditis as judged by ausculation or as estimated by exercise tolerance tests. There were no facilities for electro-cardigraphic examination. The average time away from duty for all cases was 53 days. None was seen subsequently with a relapse, or evidence of myocardial or endocardial damage.

The criticism that cardiac disease may have manifested itself later in some patients is fair, as the possibilities of adequate follow-up was for obvious reasons somewhat incomplete; but, as at least half the soldiers affected were stationed in the vicinity of the hospital for the period covered, it is equally fair to assume that, in these at least our estimation of the benignancy of their diseases was correct. The 3 cases who relapsed were as a precautionary measure downgraded for three months, as well as 5 other soldiers who persisted in complaining of residual rheumatic pain in spite of a normal B.S.R. and the absence of abnormal clinical findings.

POSSIBLE EXCITING AND PREDISPOSING FACTORS

Eight followed acute streptococcal tonsillitis with incubation periods from 2 to 10 days. Other predisposing factors were malaria (6), dysentery (4), sandfly fever (3), but as the intervening interval before the onset of polyarthritis varied from 5 days to several weeks, the significance of these is perhaps problematical. One factor was prominent in the histories of 17 cases—severe and/or unaccustomed exposure to cold or damp, often prolonged, and these patients emphatically dated their illness from this exposure. The polyarthritis began from 2 to 14 days later. The immediate antecedent history of the remaining 9 cases failed to disclose any obvious cause. Six patients gave a history of " rheumatic fever " in childhood. One man (not included in this series) exhibited the clinical picture of rheumatic fever, except for the important difference of lack of response of the arthritic symptoms to salicylates, and was found to have a small focus of infection containing gas-forming organisms. The notes of this man are as follows:

CASE 5

Aged 24. *Feb.* 12: G.S.W.R. knee—operated upon. Apparently progressing favourably. There was no history of serum having been administered. *Feb.* 27: Onset of pains in wrist, knee, and jaw. *Feb.* 28: T. 102·6°, P. 110. Healthy looking wound 6 in. long inner side of L. knee and thigh. Marked effusion into both knees. Both wrists very painful on movement; complains of pain in temporo-maxillary joints on opening mouth, movements of which are limited. Sod. sal. grs. 150 daily begun. *March* 3: T. 101·2°, P. 112. Knees as before. Marked pain in temporaro-maxillary joints. Swelling of both wrists. Pain in left shoulder and elbow. Wound explored: small abscess in adductor canal containing gas-forming organisms. *March* 9: T. normal, P. 90. Effusions have disappeared. Arthritic pains still present. *March* 20: Uninterrupted recovery; no pains.

## Discussion

A primary attack of rheumatic fever is relatively uncommon in adults. Hedley (1940) found that rheumatic fever in more than 75% of cases began before the age of 20. It is well recognized that the disease in adults does not affect the heart as frequently as in children. The 47 cases here recorded suggest that in adults, or at least in soldiers living under service conditions abroad, so-called rheumatic fever rarely causes cardiac damage. Green (1944) states: " The aetiology of rheumatic fever cannot be considered as finally solved, but it is essential that haemolytic streptococci should be placed first and foremost in all cases relating to the spread and re-activation of acute rheumatism." Most authorities agree with this. The low incidence of streptococcal infection (17%) in my cases is in itself a cogent reason for attempting to differentiate this benign syndrome. Furthermore, the incidence of these cases bore no obvious relationship to the frequency of admissions for tonsillitis to the other wards of the hospital. The most striking possible precipitating cause was severe and prolonged exposure to cold and damp (36%). It is suggested that this may in some way lower the body immunity so markedly that the unknown cause of acute rheumatism acts. To assume a latent virus infection is tempting, but so far no positive support has been given to this theory. Serum arthritis produces a picture resembling rheumatic fever making an allergic basis for all acute rheumatism attractive. Coburn's views on the hypersensitiveness of certain tissues to haemolytic streptococci are well known; in those of my cases, however, in which a bacterial or protozoan toxin might be suspected (49%), the interval between the possible cause and effect is so variable that allergy or hypersensitiveness cannot be the complete answer. The points of differentiation in the 47 cases from classical rheumatic fever are: (1) relative absence of toxaemia and malaise; (2) the quick response to treatment with salicylates; (3) the rapid fall in B.S.R.; (4) the normal red blood count—in juvenile rheumatic fever a secondary anaemia is almost a constant finding; (5) the absence of cardiac complications; (6) the infrequency of relapses or previous history; (7) the relatively insignificant rôle played by the streptococci; (8) the frequency in which a history of severe exposure to severe cold and damp is obtained. The benign nature of the disease is the chief reason for thinking that this is a clinical entity and should be differentiated from the classical type. Ferguson's designation " acute febrile polyarthritis " is favoured in order to avoid confusion with the more serious true rheumatic fever.

It must be emphasized emphatically that this article deals with a special group of males, and it is not suggested that a benign type of rheumatic fever is recognizable in children. Rheumatic fever in children and adolescents should be regarded as affecting the heart until conclusively proved otherwise.

## Summary

1. Forty-seven soldiers, all stationed abroad, suffering from a benign type of rheumatic fever are discussed.

2. The relative absence of malaise, the rapid fall in B.S.R., the normal red blood count, and the absence of cardiac complications, are the chief features in distinguishing the disease from classical rheumatic fever.

3. Streptococci infection was relatively infrequent as a precipitating cause; 36% gave a recent history of severe exposure to cold and damp.

4. The name acute febrile polyarthritis for this benign type of rheumatic fever is favoured.

## References

Copeman, W. S. C. (1944). *Ann. rheum. Dis.*, 1, 11.
Ferguson, G. C. (1943). *Canad. med. Ass. J.*, 49, 492.
Green, C. A. (1942). *Ann. rheum. Dis.*, 3, 4.
Hedley, O. F. (1940). *Publ. Hlth. Rep. Wash.*, 55, 1707.

# RHEUMATISM IN SWEDEN *

BY

W. S. C. COPEMAN

## Historical

A survey of rheumatic diseases was made by the Swedish Government at the beginning of this century, and the situation was found, as had been expected, to be that this group of diseases was involving the country in great expense, and that treatment facilities were non-existent for a large proportion of cases. In Sweden the large majority of workers are insured, and become eligible for pension based upon their previous income if they become incapacitated; rheumatic cripples thus become pensionable.

In 1915 the National Pensions Board completed an arrangement whereby a variable number of beds was reserved for these cases in general hospitals if they were unable to continue with their employment. Their fees were paid by the Board for a period up to two months.

In 1918 Kahlmeter, who was C.M.O. for the Pensions Board, conducted a further survey and found that in that year there were more than 50,000 patients pensionable from this group of diseases, whilst 5,000 new cases came into this category during the year. As a result of this the Board in 1920 bought two spas, Nynäshamn and Tranas, and built special rheumatic hospitals in these, each capable of accommodating 200 patients. A third was added in 1923 (Are). Cases were allocated to these from the central office in Stockholm.

In 1926–1927 the policy changed, and it was considered desirable that these patients should be accommodated and treated in special centres situated in the five chief regional general hospitals of the country. The advantages were considered to be: (*a*) full facilities for ancillary services and special opinions; (*b*) increased interest in academic medical circles; (*c*) facilities to train physicians for diagnosis, treatment, and research in this field.

By the end of 1927, 345 beds had been established in this way. The largest unit was at Lund (120 beds). In each case they came under the general direction of the Professor of Medicine; a physician of the rank of Docent (Reader, lecturer), with a special interest in the subject, being appointed in charge; a consulting orthopaedic surgeon, being specially attached and having beds in the same block. Two years later another such department was added in the Norrköping region.

This system worked well from the administration point of view, but conflict developed between the professors of medicine, where these were not particularly interested in the subject, and the Docent in charge, the former taking the view that rheumatism is only a minor field of general medicine and that specialized consideration would give exaggerated importance (Svartz); the Docents' view being that, with the specialized facilities and interest at their disposal, great advances could be made if the repressive academic influence could be removed, and the subject become a free speciality with the status of a special department (Edström). Some wished to revert to the special hospital system, with the addition of visiting specialists (Sunderlin) from regional centres.

## Royal Commission 1937

In 1937 a Royal Commission was appointed under the Chairmanship of Professor Höja, the President of the Board of Health, and the whole problem was discussed anew.

It was decided to provide an additional 1,200 beds, in units of 60–120, and place these units in the eight chief central county hospitals. In this way each unit would be in the centre of a region containing 150–200,000 population. The existing general hospital units were retained under the professors of medicine, but these new beds were to rank as independent departments, although the medical staff might be drawn from the professorial departments in the general hospitals. The programme provided for their completion in 5–8 years, but the occurrence of the world war, coupled with some further opposition from the professors of medicine, led to postponements. The Commission endeavoured to compromise with the professors by offering them the use of all beds not being used for rheumatic purposes for general medical overflow, but no real progress was made.

## Recent Activity and Proposals

As the result of this stasis a new and larger commission was appointed in 1941, which included representatives of the professors and the Docents.

In the summer of 1943 a member of this commission, Docent Edström, made an up-to-date survey of the present position and needs. He made a

* This Brief Report on the Incidence and Treatment of the Rheumatic Diseases in Sweden is based on a visit made in July to Malmo, Stockholm and Lund, by kind permission of the Royal Swedish Government, and Board of Health.

preliminary but exhaustive census of rheumatic diseases in four large districts—two coastal and two inland. The total population of these was 48,000, of whom he found that 2·4% had, or had had, rheumatic fever, 2% rheumatoid arthritis, 1% osteo-arthritis, and 2·6% sciatica. Other forms of non-articular rheumatic disease are not mentioned. He found, therefore, that 1/25th of the population were suffering with one of these forms of rheumatic disease at the time of his enquiry. From this it was calculated that in Sweden, with a population of 6½ millions, 100,000 individuals were permanently incapable of work, in spite of the fact that the arrangements for treating this group of diseases are probably the best in Europe.

Under the authority of the Commission, 4,000 doctors were circularized, of whom 2,000 replied, two-thirds very fully. In this way 66,500 case reports were obtained of which 5,000 were found to be duplicated (±61,000). Of these, 21,000 patients had been hospitalized for varying periods, whilst 5,000 had been too bad for this. A study was made of the length of stay of those patients who had done well, and an average minimum period of hospitalization was recommended as the result. This is as follows:

| | |
|---|---|
| Group I.—Acute rheumatic polyarthritis .. | 60 days |
| Group II.—Chr. polyarthritis (mostly rheumatoid arthritis .. .. ..<br>Group III.—Arthrosis .. .. .. .. | 90 days |
| Group IV.—Other types, including fibrositis and sciatica .. .. .. | 40 days |

To provide this accommodation for the present need would require about 3,000 additional beds, of which half would be to serve those patients at present unable to obtain any treatment; the rest, to provide for the full period of hospital stay necessary on the above calculation in order that patients do not have to be discharged prematurely from the hospitals to make room for others, as at present.

It was realized that, since research had by then started in the special units of the general hospitals, new light might be thrown on therapy in the near future which would render the provision of beds on this scale unnecessary. In view of this, it was decided to authorize the immediate provision of 2,000 of the 3,000 beds required for present purposes. It was considered that this would be sufficient to deal with Groups I and II, which constitute numerically the greater problem in Sweden.

In 1944, the Commission were satisfied that this solution of the problem was therapeutically administratively on the right lines, but the problem of after-care and rehabilitation had become urgent if these patients were to be restored to high wage-earning capacity, and this field was exhaustively explored. Their report on this has just been published.

Perhaps their most interesting recommendation, which has been approved by the Government, is the institution of "after-care beds" in every hospital treating rheumatic and orthopaedic cases, on a basis of 20% of the total beds employed in these ways. This means the provision of 20 of such beds for every 100-bedded unit. These beds are to be provided immediately, and are of a very much cheaper type than those of the main hospital, the plan (in report) suggesting that they are more in the nature of glorified huts.

When the rheumatic patient has reached the end of his time in the main hospital, and provided he is capable of looking after himself to a large extent, he will be transferred to one of these beds but will continue under the regular observation and treatment of his hospital physician. Besides saving space and increasing turnover in the main hospital, this plan is considered to condition the patients to the changed environment to which they will have to return in their homes on completion of treatment —the sudden transition from complete hospitalization to home being thus avoided.

Previously patients who did not seem susceptible to much improvement, but who needed further treatment, on discharge from a general hospital unit were transferred to hospitals for chronic disease which are situated in each country district. These cases, being beyond the range of influence of the general hospitals, tended to become neglected. Recently these have been moved and regrouped at the Regional Medical Centres. Since this was done the improvement or cure rate has gone up by 20%—mostly in the field of rheumatic diseases—with a corresponding saving of public funds. The Commission therefore recommend that these beds be also extended by 400 for the rehabilitation of long-term rheumatic cases.

The proposed total increase in the beds to be allocated for the rheumatic diseases in Sweden is therefore 400 in Class A (general hospitals), and 600 in the 8 Regional Centres, where the physician in charge will be independent of the Professor of Medicine. Such independent units will then average 120 beds each with a whole-time or nearly whole-time specialist physician in charge. In view of the size of these units under these proposals, the Professors of Medicine have realized that they would not have been able to accept responsibility for them in addition to their present commitments, and have partially withdrawn their previous objections.

* * *

The above is the outline of the Central Government's scheme, but as the provincial governments are largely autonomous in health matters it is left to them to start on whatever part of the scheme which seems to them most immediately advantageous. The Central Government will pay for the whole cost of building and half the cost of the first years' running expenses. After that, finance has to be agreed between the Provincial and the Central Government. Patients who are able to do so are expected to pay three Kronor per diem under this scheme, of which the full cost is estimated at 12 Kronor.

It is realized by the Board of Health that this extension of their programme for dealing with rheumatic diseases will involve an increase in the number of physicians specializing in this group of diseases.

Their preliminary view is that this should involve 2 years (minimum) postgraduate work in general medicine, one year as assistant in an orthopaedic department, and a further year in a physiotherapy department, spa hospital, or special department for rheumatism. In addition they consider that 6 months' study of the subject abroad should be insisted upon wherever possible.

As an indication of the degree of interest in this field of disease in Sweden it may be mentioned that the public were invited to contribute to a special fund on the occasion of King Gustav's 70th and 80th birthdays, and that the whole proceeds of the former were allocated for cancer, whilst the proceeds of the latter have been allocated for extension of treatment facilities, hospital beds, and a research institution (at the Karolinska Hospital) for the rheumatic diseases. This fund amounted to 6,500,000 Kronor. A rheumatism society under government patronage with branches in all parts of the country is being founded to maintain this interest.

(It is proposed to allocate the next fund, if the King reaches his 90th birthday, to the psychoneurotic diseases.)

# BOOK REVIEWS

*Arthritis and Allied Conditions.* By Bernard I. Comroe, M.D. Third Edition, revised and enlarged, pp. 1359. Price 60*s*. Henry Kimpton.

For a work of this magnitude to have reached a third edition within five years is the best evidence of its value and the place it has gained in the literature of the rheumatic diseases. It has been very thoroughly revised and brought abreast of the most modern knowledge of the conditions dealt with, which comprise every aspect of rheumatology. Many new chapters have been added on recent advances in diagnosis and treatment, embracing further experience with gold therapy, and also with the sulphonamides and penicillin. One of the most valuable of these is an introductory chapter providing a diagnostic digest of the average arthritic problem for the general practitioner, consideration of whose needs is a feature of the whole book. The technique and indications for massage receive special attention, with some excellent illustrations of methods; physiotherapy, occupational therapy, spas, climate, psychogenic factors, and various modes of treatment are fully described and their indications set forth in a clear and attractive manner. The practitioner will find the chapters on the painful shoulder, painful feet, and backache helpful in tackling the many difficulties presenting themselves in everyday practice.

A striking and interesting chapter is that entitled " Mistakes in the Diagnosis and Handling of Patients with Arthritis and Allied Conditions ": the author's enumeration of 238 mistakes furnishes serious matter for thought for all concerned with the treatment of rheumatism. It would be interesting to quote from them at length, but more profitable to read the chapter itself. One, however, must be quoted in full on account of its bearing on the work of the Empire Rheumatism Council: " Mistake 63 is the lack of sufficient funds for investigative work and lack of adequate hospital facilities for the care of arthritic patients in this country [the U.S.A.]. There are not more than 200 free beds in the United States for the care of rheumatic patients as contrasted with 100,000 free beds for the care of tuberculous patients, despite the fact that arthritis and allied conditions are more common than the sum of all tuberculosis, cancer, diabetes and heart disease." While it may be claimed that in Great Britain a much larger number of beds for the arthritic are available, they are far short of what is required if this great problem, which affects so seriously the working capacity of the country, is to be properly dealt with.

This book should find a place on the shelves of all who have to deal with rheumatic diseases, and its careful study will lead the general practitioner to find in his rheumatic patients a source of much interest instead of boredom, as well as adding greatly to his resources in their treatment.

*Arthritis. What can be done about it.* Alfred E. Phelps, M.D. Pp. 90, 6*s*. Medical Publications, Ltd. London.

This little book is designed as stated on the cover to tell how the patient may co-operate with his doctor to help speed effective treatment. It is written by a physician of wide experience in private practice who has done much work on arthritis, and sets forth in clear and simple language information which cannot fail to be helpful to sufferers from arthritis in its various forms and may even be found practical and instructive by the doctor also. The various factors in causation are clearly explained, with stress on those which are in the power of the patient to influence; but at the same time the author describes in simple terms the part played by infective factors and the more common bacteria believed to be concerned in aetiology. Diet, rest and exercise, the influence of posture, strain, trauma, climate and other factors are discussed. Modern methods of treatment are explained, such as gold and vaccines, though the author wisely says that of all methods of treating arthritis vaccine therapy is the most difficult; the dosage of gold which is mentioned is higher than is necessary, and it should be stressed in any future edition that greater safety lies in smaller doses than were formerly the vogue and the effects are likely to be quite as good. The book can be cordially recommended for the purpose for which it has been written.

# EMPIRE RHEUMATISM COUNCIL

## ANNUAL REPORT, 1945 *

BY

LORD HORDER

MY LORDS, LADIES, AND GENTLEMEN,

It is with sincere pleasure that I once more welcome members of the Empire Rheumatism Council assembled in Annual Meeting. Since November 1938 your officers have been deprived of the valued assistance which you can give them by debate on the records of the past and the programme of the future. Between then and now you have been informed of all important developments through the Annual Reports, circulated with invitations to submit criticisms and—if any members thought that necessary—to ask for the summoning of a meeting. That procedure—though it brought each year a tacit vote of confidence by the absence of criticisms (a negative gesture which is gratefully acknowledged)—could not be as satisfactory as the stimulus to be drawn from discussions with members. But, combined with the "telescoping" of all the Standing Committees into one fully representative War Emergency Committee, it was the only practicable way of carrying on during the war emergency.

In 1939 there was a not inconsiderable body of opinion among those whom you had elected to the control of the Council that its work should be suspended for the duration of the war in Europe. The ultimate decision to continue without interruption, but with all possible economy of administration, was, after discussion, accepted unanimously. The record of accomplishment since that time, despite obstacles, suggests that the decision was a wise one. True, the highly promising progress of 1937 was not maintained. The 1938 Annual Report, as you will recall, noted "serious anxiety" regarding the international position: our opening public appeal meeting at the Mansion House (1939) when our Royal President, in an eloquent address, "with all confidence" commended to the good will of the people the work of our Council, actually coincided with the date of the first War Budget, with its implicit call to earmark all national resources for the defence of civilization. The Fates had granted us only one year of peaceful progress.

### WAR-TIME DIFFICULTIES

War-time difficulties were great. The chief of these was the consequent diversion of scientific workers from our campaign. Those with special knowledge of the problems of rheumatic diseases were, in 1939, few in number. Most of them were withdrawn for the Defence Services and for the Emergency Medical Service. Of the small remnant left practically all accepted many extra duties in the national interest. Thus for the scientific work of our Council there was left, not the leisure hours of a few—for leisure hours simply did not exist—but the resolution of that few to add to an already overburdened life further effort for a good cause. To them, especially to the serving officers who gave up much of their scanty periods of leave to carry on our work, we owe a great debt of gratitude.

Damage from enemy action was not inconsiderable. The office of the Council had to be vacated when the building had been almost completely destroyed after a series of bombing attacks; fortunately all records were saved. The temporary office—there was no removal to a "safe area"—was slightly damaged and for a long period threatened by an unexploded bomb. But work was never suspended. A bomb destroyed the laboratory on the premises of the St. John Clinic; the equipment was saved. A more serious "casualty" was the taking over by the E.M.S. of the Rheumatic Unit—a laboratory and an in-patient ward—established at the Hospital of St. John and St. Elizabeth. The laboratory used by the Naval Research Foundation at Greenwich was closed for a short period but subsequently, by agreement with the Admiralty Medical Services, transferred to a site near Bristol. From 1940 only one of our three established laboratories was working.

Research progress suffered also from some frustrated hopes. I shall cite only one example. Some scientists of Warsaw University were announced to have made a notable advance in discovering a serological test of diagnostic value in rheumatic disease. We were in touch with them and were on the point of welcoming one of them to England for a full test here when the Nazis engulfed Poland. All efforts have failed to trace any one of the group as a refugee. We are forced to conclude that the Polish scientists were among the many victims of the Nazi policy of exterminating the intellectual leaders of the peoples in occupied countries.

Interruption of our work in promoting the establishment of treatment centres was also serious. In 1939 arrangements had been completed for the

* The following is the text of the Report presented by Lord Horder for approval to the Annual Meeting of the Empire Rheumatism Council.

establishment at a group of collieries of a model treatment centre, the working experience of which would have been a guide to the whole coal-mining industry. War controls stopped the building of the Centre. Several other promising developments of treatment centres had to be postponed for cognate reasons. Fortunately, the rheumatism department promoted by the Council at the West London Hospital developed its activities considerably. Other treatment centres in the kingdom which are in close relation with our Council suffered severely.

Another war check to the prospects of rheumatic patients obtaining relief may be recorded. Before the war we had an assurance from the London County Council Medical Services that the admitted serious lack of rheumatism in-patient accommodation in the area under their administration would be partly remedied by the ear-marking of beds released by the dwindling incidence of tuberculosis. Unhappily the war, causing a demand for beds for battle casualties and also causing a recrudescence of tuberculosis, has nullified this. Thus, whilst in 1939 there was a growth of facilities for treatment, the position in 1945 is worse than it was six years ago, and calls for the most energetic action lest the ravages of rheumatic disease—the most serious enemy of home happiness and industrial efficiency —should be increased.

One further frustration is to be noted. Negotiations with friends in Australia had led to the summoning there in 1940 of a Continental Medical Congress the chief work of which would have been the consideration of the problems of rheumatism and the establishment of a council affiliated with our own. The war made postponement necessary. This delay in recruiting a whole continent to the war on rheumatism was a great disappointment.

This review of obstacles is not made by way of vain lament but to indicate the difficulties encountered and to impress the need for vigorous effort in the future.

To pass now to the record of achievement. It will be convenient to summarize briefly the work of the whole period, under its chief headings, since the foundation of the Council.

## Research

The chief work of the Council has been in the direction of laboratory and clinical research into causes and means of treatment. Owing to the very generous co-operation of the Press (individual newspapers and agencies) our existence and our aims have had world-wide attention. Correspondence has totalled thousands of letters, some from remote quarters of the globe. A proportion of these, embodying crank notions or having palpably a money motive, called for no other attention than a civil reply. We could not enter into investigations, for example, as to the relations of the measurements of the Pyramids to rheumatic disease; nor attempt to prove or disprove the claim that a certain variety of apple (grown by the correspondent) was an infallible cure for all forms of rheumatism. Nor were we responsive to a number of domestic and foreign "healers," without any traceable medical qualifications, who would be willing to come to London (if provided with large sums of money) and prove conclusively the efficacy of their "cures." But no suggestion which was of good intent was ignored, whether from lay or medical sources, and, in addition to the many suggestions which could be submitted at once to laboratory or clinical investigation, there remain on record several which were impossible of investigation during the lack of a national chain of treatment centres dealing with the great body of rheumatic sufferers. These will be studied in the future when that lack is remedied.

Whilst only an insignificant percentage of sufferers have available, under observed conditions, those means of treatment which the present state of medical knowledge affords, clinical research is seriously handicapped in securing definite decisions. The right verdict on any proposed new treatment, or new development of any existing method of treatment, can be arrived at only by observation, under conditions of control, of a large number of patients accurately diagnosed in the first instance and subsequently checked for a period to note whether "cure" or substantial alleviation is permanent or merely temporary. There is often a temptation to claim a "cure" by some individual practitioner founded on his own experience only. But it is a principle of medical science to define the word "cure" as something which can be applied by the medical profession generally.

In this field of clinical research our scientific committees have dealt with 256 suggestions from many parts of the world. Most of these required only brief attention because they were repetitions of, or slight variants from, well-known treatments. Others required, and received, more particular study. Grateful acknowledgement is due to the few available treatment centres of the kingdom for their cordial co-operation in conducting tests.

In laboratory research we have financed five long-term investigations for periods ranging up to eight years and fifteen short-term investigations, usually for a term of one year. These have been at universities and hospitals possessing the necessary facilities. A major task has been that of the Naval Research Foundation. This was founded in 1938 to investigate the causes, and the best means of treatment, of rheumatic disease in the Training Establishments of the Royal Navy. Funds were provided by the generosity of Mr. Frederick Pearson and the Sir Halley Stewart Trust. The work of the Foundation has been continued until the present year; on a reduced scale during the past two years. The Admiralty Medical Services have judged it to be highly satisfactory. It was the subject of a thesis by Dr. C. A. Green, Director of the Foundation, to Edinburgh University (1941), gaining for him the M.D. and Ph.D. degrees and a gold medal. A branch of the Foundation was established at the laboratories of the Frederick Pearson Foundation at the West London Hospital, and work there sug-

gested a new line of biochemical investigation, which, for its proper examination, must await the establishment of the national chain of treatment centres.

A munificent gift by Sir Alexander Maclean financed the establishment in 1937 of two research laboratories. That at the Hospital of St. John and St. Elizabeth, with its in-patient ward, called for an added block to the hospital premises, the foundation stone of which was laid by the late Sir Kingsley Wood, then Minister of Health. This, as before noted, was closed in 1939 owing to the hospital being taken over by the War Emergency Service. The Research Director, Dr. C. B. Dyson, had done valuable work resulting in the suggestion of a virus origin for certain infective forms of rheumatic disease. Dr. Dyson has since continued this work as far as practicable in County Durham. Another piece of research was undertaken at this hospital by Dr. Hugh Burt, on the value of occupational therapy in the treatment of rheumatic disease. This was a pioneer effort in that field and Dr. Burt's conclusions have since been widely adopted.

The second laboratory was established at the St. John Clinic, London, having a link with the St. Stephen's Hospital Rheumatism Ward. The Director, Dr. H. J. Taylor, did valuable work, especially on the physical problems of rheumatic disease. The laboratory was destroyed by enemy action in October 1940.

It would be impossible in this report to attempt even the briefest summary of the results of our laboratory research work. The official journal, *Annals of the Rheumatic Diseases*, has recorded them fully. I commend this publication to members of the Council and to others who are interested in public health questions. Our cordial thanks are due to the British Editors and their colleagues in the United States who have maintained the journal during six most difficult years. To the best of my knowledge it is the only European scientific publication dealing with rheumatism which has survived war conditions. As you have been informed, it is now published quarterly by the British Medical Association, with an Editorial Board representing the Empire Rheumatism Council and the *British Medical Journal*.

I venture the opinion, which is endorsed by scientific observers abroad, that, regarded in the aggregate, our research work has contributed materially to existing knowledge of the problems of rheumatism. That negative, or "not-as-yet-proven," decisions have resulted on some questions was to be expected. To rheumatic sufferers this is disappointing; the scientist will recognize that even a negative decision has its value and that it would be a failure in duty to neglect to investigate any suggestion which seemed to offer hope and without regard to the issue.

### Provision of Treatment

It is in this section of our work that the most definite progress may be recorded. The decision of the Government, endorsed by Parliament, that in future health policy all those suffering from sickness or injury would have available "treatment appropriate to their needs" opened a new era in the history of the social plague of rheumatism. Though there was in the announcement of the decision no specific mention of rheumatic disease (nor of several other diseases) the governing word was "all," and specific statements since then have cleared away any possible suspicion that "all" meant "all except the rheumatics." We must give the credit for this promise of great reform in health administration to the wisdom of our rulers, but the Empire Rheumatism Council fairly claims its share of that credit in arousing public opinion, the ultimate authority in a democratic community.

It will be useful to note, very briefly, the progress of events leading to this decision:

1. The National Health Insurance Act of 1911 provided medical advice and treatment for employed persons below a certain level of remuneration. There were, however, two great defects. The wage-earner was insured but not his or her dependants. Sufferers from rheumatic disease were in the main excluded from appropriate treatment, though it was implicitly recognized that for them something more was needed than would be available to the panel practitioner. So provision was made in a subsequent amending Act for rheumatism treatment as an "additional benefit" and for the approval of treatment centres to give that treatment. But two limitations were imposed which debarred most sufferers. The "additional benefit" was available only to those belonging to the prosperous Approved Societies which had surplus funds after meeting other obligations; and treatment centres (approved) were insignificant in number and out of reach of the majority of the population.

2. At the beginning of its campaign the Empire Rheumatism Council recognized that the treatment problem was of paramount importance, since success attending research into aetiological causes might not be achieved for many years. There was one certain factor in facing this problem: that many morbid conditions are successfully treated in medical practice though their aetiological cause is not yet known. There were two factors not so certain: (*a*) whether, in the present state of medical knowledge and given proper facilities, rheumatism could be successfully treated, and (*b*) whether, in our country, knowledge on the subject needed to be supplemented to make it as good as in other countries. Prompt steps were taken to get more information on these points by a survey of rheumatism treatment at home, in Western Europe and in North America. Two research workers, Dr. W. S. Tegner and Dr. Duthie, were engaged on this task, the former covering most of Western and Central Europe and some centres in North America, the latter some centres in the United States. A generous gift from Sir Alexander Walker provided the expenses of Dr. Tegner's investigations. The conclusions from this survey were: as regards (*a*) that treatment based on present knowledge was efficacious

in the majority of cases, if applied at an early stage, but in a progressively reducing proportion of cases if neglected; as regards (*b*) that British knowledge of treatment was fully up to the international standard but that its application was much below the level in some other countries.

3. The result of this survey and of much other ascertainment was the publication (March 1941) of " Rheumatism—A Plan for National Action." This was submitted, in the first instance, as a tentative plan with an invitation for constructive criticism both on its medical and on its administrative aspects. No such criticism came, but, on the contrary, full approval from the medical and lay press, from public health committees in different quarters of the kingdom, and from several countries abroad. The Plan sought to show how this social plague could be combated with reasonable regard to economy and to our national way of life. It may be confidently assumed that, in its main principles at least, the Plan will be the basis of the future national action recently promised.

4. In October 1941 came the Minister of Health's statement of governmental policy to the effect that a post-war scheme would ensure " that everyone will receive the treatment appropriate to his need."

5. A post-war survey of treatment in Sweden, carried out (1945) by Dr. W. S. C. Copeman with the courteous assistance of the Swedish Government, fully confirmed the conclusions stated in paragraph 2.*

The promise of national action leaves to the Empire Rheumatism Council for the future the task of doing its utmost to ensure that the treatment centres established will have efficient medical staffing.

On the subject of treatment there may be added to this survey a few additional facts. For the civilians' benefit the Treatment Centre at the West London Hospital has done constantly progressing work; other centres have been assisted to the best of our ability; and by private correspondence, amounting in the aggregate to several hundred letters each year, individual sufferers have been advised. For the benefit of the Armed Services we have acted in close and efficacious co-operation with the Naval Medical Services; in the Army we have secured the establishment in one Home Command of a special rheumatism ward and another in France, where Dr. W. S. C. Copeman on the one hand set an example of good results following improvised equipment, and on the other hand showed that economy can be consistent with efficiency. (See the *Journal of the R.A.M.C.*, May 1940.)

## Education

A very important need of the near future is to ensure that a National Plan of Treatment is not hampered in its development by lack of practitioners to staff effectively the new centres to be set up. On the long-term view this matter of educating the profession must be the task, primarily, of the University Medical Teaching Schools and our part is to stimulate this. It is encouraging to note that Bristol University and the West London Hospital have set a good example. In the short-term view it is clear that there is immediate need for a comprehensive series of postgraduate courses to give a selected number of practitioners special training in the diagnosis and treatment of rheumatic disease. The necessity for this has been recognized for some time. Action was hindered by war conditions making it impossible to obtain both teachers and learners. In preparation for more favourable conditions a representative Committee was set up (1944) with Sir Adolphe Abrahams as its Chairman and including representatives of other medical bodies such as the British Orthopaedic Association, the British Association of Physical Medicine, and the Society of Industrial Health Officers. Clearly this work must not be restricted to a London centre. But a beginning has been made this autumn and extension of the work will have a high priority in 1946.

Not only education of practitioners but education of the public—its leaders and the general commonalty—on the prime importance of checking the ravages of rheumatism, is also our concern. In this section of work we have reason to be well satisfied so far. Public opinion, which, I repeat, is the controlling force of official action, has been effectively aroused. No longer is rheumatism regarded as a misfortune inevitable because of our climate, nor as something which is comparatively trifling in the category of the ills of mankind. There have been published large numbers of articles on the subject in the medical press and in the important lay publications of the British Commonwealth. I cannot attempt a comparative statement of the meagre press attention to rheumatism before 1938 and since, but the growth has been from an insignificant amount to its present impressive volume. The generosity of the press has been all the more significant in face of the grave difficulties from paper shortage.

Educational meetings have been rare in the past year and indeed in all the war years. In pre-war days they averaged more than one per week: the Rotary movement had been very helpful in this respect, providing audiences for over sixty addresses in London and the provinces.

## Administration

Cordial relations have been maintained with the Ministry of Health, the Medical Research Council, and other public and private organizatons within the Empire and with our colleagues in the United States. The Minister of Health has taken an important step in setting up a rheumatism subcommittee of his Medical Advisory Committee. The Chairman of this sub-committee is Professor Henry Cohen of Liverpool University, and your Council is well represented upon it.

You will have occasion later to express your thanks to the War Emergency Committee and to the staff. I shall not anticipate that nor shall I trespass

* See page 17.

on the report of the Finance Committee, which will review our financial position, but I venture an approximate cost analysis of the proportion of expenditure in the respective departments of our work. Such an analysis shows 38% of the total was spent on research work; 36% on treatment work; 15% on educational propaganda, and the remaining 11% roughly divided between appeal work and general administration. Exact detailed figures of our wide range of tasks would be difficult owing to the fact that one official, with one clerical assistant, acts as appeals secretary, financial secretary, publicity officer, and general administrator.

An agreement for full co-operation with the Heberden Society, a group of medical men devoted to the discussion of the clinical problems of rheumatic disease, will lead to a valuable development of our campaign.

The committees which you elect at this meeting will have available to them a series of memoranda on various pending developments, scientific and administrative. Detailed discussion on these matters may well be left to their deliberations. One step had to be taken by the War Emergency Committee in advance—viz. the transfer of the headquarters office from its temporary site in Hampstead to offices at B.M.A. House, Tavistock Square, leased from the Royal Medical Benevolent Fund Ladies' Guild.

Our committees and their officers will face strenuous and difficult tasks, but I think they will find sustaining comfort in the record of achievement since our foundation which I have attempted to give you. A scheme of national treatment has been promised on the highest authority. Public opinion has been definitely recruited for our support. A good measure of progress has been achieved in research.

### The Future

If I were to state what I consider to be our tasks in the coming year and in future years I should say they are:

1. To continue research, laboratory and clinical, inviting proposals from all competent quarters.
2. To promote measures for postgraduate and graduate education in the problems of rheumatism.
3. By lectures and articles to keep public opinion instructed.
4. To resume the interrupted effort to establish affiliated Councils in the provinces and the Overseas Dominions.

For the future I have no misgivings. As I was never tempted to join those happy optimists who thought that the cease-fire order would promptly usher in a rosy dawn of days when we might " live and lie reclined " as in the Land of the Lotus-Eaters, so I am convinced that austere years will follow. Yet I have great faith in the stubborn courage and the good sense of the British race to face difficult times. That faith is supported by the experience of many crises in our history when quick recovery from what threatened to be mortal wounds was followed by great advances in prosperity. It is above all the good sense of the people that will need to be fully evoked for the immediate future—the good sense to insist that the well-being of its people is the surest basis of a nation's security and that the most tragic form of economic waste is the neglect of preventable disease.

# CHRONIC RHEUMATISM IN SCOTLAND

The Department of Health for Scotland has recently issued a report on chronic rheumatic disease drawn up by the Scottish Medical Advisory Committee. It is an admirably lucid and comprehensive review, and will rank with the key publications on this subject. The authors are Sir John Fraser, Sir Alexander S. M. Macgregor, Professor Adam Patrick, Dr. D. Dale Logan, Dr. A. F. Wilkie Millar, Dr. J. M. Johnston and Mr. H. V. de Lorey. The publishers are H.M. Stationery Office, Edinburgh, and the price sixpence.

The authors begin with stating the classification they have adopted, which, though not as detailed as other classifications such as that of the Royal College of Physicians (England), is sufficiently clear for their purpose. (The " acute group," rheumatic fever and sub-acute rheumatism, are excluded, as also is arthritis of known specific aetiology—e.g. gonococcal arthritis.) Conclusions as to incidence are drawn from a wide range of statistical evidence, chiefly the records of the Scottish N.H.I. There is the wise precaution of noting that the figures are subject to the qualification that many conditions with insufficient care in diagnosis may be mistaken for rheumatism. Throughout the authors impress that accurate diagnosis at an early stage is of the first importance in any national plan for the treatment of rheumatic sufferers. " Ineffective treatment is often due to insufficient clinical investigation at the outset."

The statistics of incidence, allowing for all reservations, give an impressive picture of the damage to home happiness and industrial efficiency from the ravages of rheumatic disease. In Scotland (population 5,000,000) among the insured class in 1938 there were 45,300 new cases of incapacity from this cause; and the insured class by no means represents the whole of the nation. Occupational figures of incidence are highly interesting. " It may be estimated in round numbers that each year rheumatic conditions incapacitate:—

*Men.*—1 in every 21 miners.
1 ,, ,, 29 general labourers.
1 ,, ,, 37 transport workers.
1 ,, ,, 39 metal workers (including iron and steel industries).

*Women.*—1 in every 37 domestic workers (including charwomen).
1 ,, ,, 37 agricultural and fishing workers.
1 ,, ,, 40 outdoor workers.
1 ,, ,, 43 transport workers."

To deal with this serious incidence it is recorded that " there is general agreement that the existing facilities in Scotland for the diagnosis and treatment of rheumatism are quite inadequate. For economic reasons the great majority of rheumatic patients depend on the services of the general practitioners, the general hospitals and a few small special clinics. The resources of most nursing homes and the private services of skilled physiotherapists are limited and are beyond the means of all but a few; the same is true of the Scottish Spas and the one hydropathic institution which has some equipment and arrangements for medical supervision. Most rheumatic patients usually need some form or other of physiotherapy which can be given by the general practitioner only " if he possesses the necessary apparatus and assistance for systematic treatment. Treatment by the family doctor, therefore, is necessarily limited in most instances to symptomatic relief in the more acute phases of chronic rhematism and to a few forms of general and local medication."

So much for the evil. As to means of relief the authors state:—

" So little is known about the nature and cause of the rheumatic diseases that treatment is still largely empirical. Nevertheless, if begun before the disease is far advanced, therapeutic measures can do much to alleviate symptoms, lessen the risk of deformity and cut short the period of disablement. The Sub-committee were impressed by the encouraging results obtained by thorough treatment given under proper conditions."

It is a cautious estimate of the prospects of relief, distinctly below what may be styled in mining terms the " battery reports " of well-administered treatment centres. But it is sufficient to lead the Rheumatism Sub-committee to strong recommendations:—

" We recommend that, in at least one hospital affiliated to each of the four Universities, from 20 to 40 beds should be available as a central unit for the intensive study of chronic rheumatism, especially, to begin with, of the articular types. These central units might be linked up with the University Departments of Medicine and Therapeutics, a close liaison being maintained with the orthopaedic department of the hospital, the service being under the direction of a responsible physician. The primary functions of the central units should be to provide for the full clinical investigation of patients over a period sufficient to determine the general lines of treatment. In addition the central units would provide for under-graduate and post-graduate instruction, and special training for young physicians in the problems of rheumatism."

Concerning the value of peripheral local clinics linked with those Centres the report is not so emphatic, but concludes:—

"After critical appraisement of the problems involved, we are of the opinion that there is a definite place for

peripheral clinics, provided that a start is made on modest lines. In accordance with the principle that the subject of rheumatism ought not to be divorced from general medicine by the creation of a narrow speciality, we are definitely opposed to the establishment of clinics designed solely for the treatment of rheumatism. As many forms of treatment are common to rheumatic and orthopaedic conditions, we advise that from the outset the rheumatism service should be associated with the orthopaedic service and therefore a suitable starting-point is already provided in the orthopaedic clinics in various parts of Scotland. Careful attention must be given to the question of staff. The creation of large numbers of rheumatism specialists should be avoided but, on the other hand, those in charge of treatment should have a special knowledge of rheumatic diseases. So long as regular visits are made to peripheral clinics by the specialists " from the central units, there is no reason why a local general practitioner, if suitably qualified, should not be in charge of a clinic. We envisage a two-way contact between the family doctor and the specialists with the object of ensuring the close supervision of the treatment of patients."

The importance of physiotherapy is fully recognized (and with it, occupational therapy and hydrotherapy). But the authors are critical in observing that there is danger of a physical treatment becoming a matter of routine. The unimportance of expensive apparatus is emphasized. There is good reference to the use of medicaments and to what may be termed the " promotive causes " of rheumatic disease and a valuable suggestion of " the urgent need for the co-ordination of research in industrial medicine with the investigations of the clinicians and the laboratory worker."

The report may be warmly commended to the study of all who are interested in the problems of rheumatism.